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Intergenerational Education Persistence: Evidence from Molecular Genetic Data

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Abstract

This paper exploits molecular genetic data to quantify genetic confounding in parent-child educational outcomes. We develop a model of the intergenerational transmission of education based on insights from the literature on social science genetics. The model distinguishes between two types of genetic confounding. First, narrow genetic confounding reflects the direct transmission of genetic predisposition towards education. Second, broad genetic confounding captures direct genetic transmission as well as genetic nurture, i.e., an influence of parental genes on children's outcome through the family environment. Next, we use the Avon Longitudinal Study of Parents and Children (ALSPAC) data to decompose the association between parental years of education and their offspring's grades on Key Stage 4 national exams. To proxy genetic endowments, we construct Educational Attainment Polygenic Indices (EA PGIs) for parents and children. To correct for measurement error, we use Obviously-Related Instrumental Variables (ORIV) based on two independent PGIs. The results suggest that 'broad genetic confounding' explains 30-45% of the parent-child educational association, and 'narrow genetic confounding' 18-33%. We find no meaningful differences between mothers and fathers. Using our model, we compare our estimates to twin and adoptee designs, and show how molecular genetic approaches can recover both broad and narrow genetic confounding under plausibly weaker assumptions and with arguably greater external validity.

Keywords: ALSPAC, Education, Intergenerational Mobility, Polygenic Index, Genetic endowments

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1 Introduction

Educational attainment improves your life chances: it leads to higher wages (e.g., Card, 1999), and it is linked to better health and other non-monetary benefits (e.g., Oreopoulos and Salvanes, 2011; Galama et al., 2018). One of its most important predictors is parental educational attainment (e.g., Haveman and Wolfe, 1995; Van der Weide et al., 2021; OECD, 2018). Ideally, one's life chances should not depend on parental outcomes since a strong intergenerational association may reflect a lack of educational opportunities for children from less advantaged backgrounds. As such, the intergenerational association in educational attainment is an important barometer for the equity and efficiency of a society (e.g., Corak, 2013; Mogstad and Torsvik, 2022).

Parents transmit both genes as well as environmental influences onto their offspring, and therefore intergenerational associations reflect a mixed bag of genetic ('nature') and environmental effects ('nurture'). Distinguishing between environmental and genetic transmission is important from a moral, philosophical, as well as policy perspective (e.g., Black and Devereux, 2011). If the intergenerational association mainly reflects a causal effect of parental education or other environmental advantages, then this represents a widely shared violation of equality of opportunity. If, on the other hand, the intergenerational association mostly derives from inherited abilities, people have wildly different moral intuitions (see e.g., Pereira, 2021). Some may say this also constitutes an infringement of equality of opportunity (i.e., one's genes constitute circumstances beyond one's control, and therefore provide an unfair source of advantage), while others interpret it as a sign of meritocracy (i.e., one's genes reflect abilities and merit, and a strong intergenerational persistence is consistent with equality of opportunity). To engage in a meaningful moral discussion, it is essential to quantify the role of genetic transmission in the intergenerational association in educational outcomes.

From a policy perspective, if most of the intergenerational association stems from a causal effect of parental education on offspring educational outcomes, this provides a clear policy lever to reduce intergenerational persistence of advantage and enhance child development (Björklund and Salvanes, 2011). Moreover, when making educational policies, policymakers should consider externalities onto the next generation (e.g., Björklund and Salvanes, 2011; Holmlund et al., 2011). Alternatively, a strong role for genetic transmission in driving intergenerational persistence would not leave policymakers empty-handed (e.g., Goldberger, 1979), but it would change the type of response. Genetic effects on education are not an immutable biological laws, as they arise within a certain societal context (e.g., Harden, 2021). Hence, when genetic transmission is important then e.g., changes in the school environment or the labor market may be the more appropriate response if policymakers aim to enhance upward mobility (Sacerdote, 2011).

In this paper, we develop a two-generation model, building upon the frameworks of Becker and Tomes (1979) and Solon (1999), while incorporating recent insights from the social-science genetics literature (e.g., Kong et al., 2018; Okbay et al., 2022). Based on this model, we distinguish two types of genetic confounding

in the intergenerational association between parents' and children's education. *Narrow genetic confounding* covers the genetic transmission from parents to children and the direct causal effect of the transmitted genes on the child's educational attainment. *Broad genetic confounding* additionally includes the effects of parental genes that operate through the rearing environment parents provide (sometimes referred to as *genetic nurture*).[1] We use this framework to clarify which types of genetic confounding are captured by different empirical approaches commonly used in the literature, such as children-of-twins designs, adoption studies, and donor-conception studies.

Empirically, we use data from the Avon Longitudinal Study of Parents and their Children (ALSPAC), a British cohort born in the Avon region between 1991 and 1993. The original sample size contains over 10,000 mother-child pairs, while genetic data was available for about 1,500 fathers. We extend our sample size by imputing the genome of fathers based on the mother and offspring genome following the method proposed by Young et al. (2022). This leads to a final sample size of 4,032 parent-offspring trios. We exploit the existence of molecular genetic data to decompose the intergenerational association into narrow and broad genetic components.

We observe a relatively strong association between parental education and their offspring's test scores. Specifically, each additional year of schooling completed by the mother (father) is associated with an increase of approximately 14 (12) points in the child's Key Stage 4 test score, relative to a mean of 437 — roughly a 3 percent increase. In turn, we augment the basic intergenerational regression model by incorporating the parental educational attainment polygenic indices (EA PGI). In theory, if measured without error, the parental EA PGIs account for both direct genetic transmission and genetic nurture ('broad genetic confounding') such that the remaining association between parental education and the child's test score would reflect environmental influences.[2] However, given that existing PGIs are subject to measurement error, we employ Obviously-Related Instrumental Variables (ORIV; Gillen et al., 2019; van Kippersluis et al., 2023) using two PGIs trained on two independent training samples.

Our findings suggest that broad genetic confounding explains 32-45% of the mother-child association, and 29-41% of the father-child association. The results further show that narrow genetic confounding explains between 18% and 33% of the parent-child education association, or about 56-73% of total genetic confounding. Hence, between one-third to one-half of the intergenerational association in educational outcomes stems from broad genetic confounding, with both direct genetic inheritance as well as genetic nurture each playing a role. Still, both maternal and paternal education remain important after correcting for genetic confounding, and there are no meaningful differences between mothers and fathers. This remaining association between parental

[1] We focus on intergenerational transmission of education. See Solon (1999) and Eshaghnia et al. (2022) for an overview of the literature on intergenerational mobility in income and other socioeconomic indicators. See Björklund and Salvanes (2011) and Heckman and Mosso (2014) for reviews of the role of families on child development.

[2] Even if the EA PGI were measured without error, it only captures additive and common genetic variation, excluding gene-gene interactions and rare genetic variants. There is however strong evidence for additive (i.e., non-dominant) effects of single SNPs (Okbay et al., 2022), and the variance explained by rare coding variants is much smaller than that explained by PGIs (Chen et al., 2023).

education and offspring educational outcomes does not necessarily represent a causal effect, as it also contains other environmental correlates of parental education. Whereas the limited sample size does not enable us to provide conclusive evidence, auxiliary analyses using the British Raising of School-Leaving Age (RoSLA) reform following Dickson et al. (2016) suggest that at least part of the remaining environmental association derives from a causal effect of parental education on offspring test scores.

Studies over the past decades have used innovative designs to decompose intergenerational associations into nature and nurture (see e.g., Holmlund et al., 2011; Björklund and Jäntti, 2020; Mogstad and Torsvik, 2022, for recent reviews). In Children of Twin (CoT) designs, one explores the degree to which offspring from monozygotic twin parents have reached different levels of education (e.g., Björklund and Salvanes, 2011; Baier et al., 2022). As we will show below, this approach controls for broad genetic confounding. Second, adoption designs with random assignment of the adoptee, or info on both adoption and biological parents, are popular to separate environmental from genetic influences of parents on children (see e.g., Björklund et al., 2006; Sacerdote, 2007; Black et al., 2020; Fagereng et al., 2021). In the same spirit, donor-conceived children were used to separate environmental from genetic effects (Rasmussen et al., 2024). By comparing parent-child associations among adopted (or donor-conceived) children versus biological children, these studies can isolate narrow genetic confounding (direct genetic transmission).[3]

Compared to these traditional methods, our approach relies on arguably weaker assumptions and, provided that appropriate genetic data are available, enables the estimation of both forms of genetic confounding within a single sample. Using molecular genetic data also avoids the reliance on rare cases and small samples, such as adoptees, donor-conceived individuals, or children of twins. Overall, we argue that combining molecular genetic data offers a structured way to disentangle genetic and environmental contributions to intergenerational transmission, as well as enabling the estimation of the relative roles of narrow and broad genetic confounding.

The most closely related papers are Isungset et al. (2022), Rustichini et al. (2024), Fletcher et al. (2023), and van Alten et al. (2025).[4] Isungset et al. (2022) adopt a somewhat similar approach using the Norwegian MoBa data and estimate that 15-18% of the parent-child association is due to genetic transmission with no meaningful differences between mothers and fathers. There are two differences to our approach. First, Isungset et al. (2022) control for the *child's* PGI instead of the *parental* PGIs to account for genetic transmission.[5] As we argue below, controlling for the child's PGI only captures a limited amount of genetic transmission, and delivers

[3] A third strand of literature employs instrumental variable (IV) strategies to isolate variation in parental education that is independent of genetic endowments (see Holmlund et al., 2011; Björklund and Jäntti, 2012; Mogstad and Torsvik, 2022, for overviews). While these studies effectively identify the causal effect of parental education on offspring educational outcomes, the estimated treatment effects are local and typically account for only a small share of the intergenerational association (e.g., Black et al., 2005; Holmlund et al., 2011; Mogstad and Torsvik, 2022). Moreover, their objective differs: IV studies do not disentangle genetic transmission from other environmental influences. Consequently, if the causal effect of parental education is small, it remains unclear whether the residual intergenerational association primarily reflects genetic factors or environmental correlates of parental education.

[4] See Conley et al. (2015); Liu (2018); Lin (2020); McGue et al. (2020); Verweij and Keizer (2022) for related papers in psychology and sociology. None of these papers account for measurement error in the PGIs.

[5] Isungset et al. (2022) do present results from a specification that includes parental education and parental PGIs as independent variables, but the purpose of this analysis is to investigate whether genetic nurture (i.e., the effect of parental PGIs on offspring outcomes) is mediated through parental education.

a parameter that is hard to interpret. Second, they do not control for measurement error in the PGI. This is not innocuous. In our estimates without corrections for measurement error, we find an attenuation of 22-26%. When using ORIV to account for measurement error, the attenuation is as large as 41-45%, suggesting that correcting for measurement error is quantitatively meaningful. Fletcher et al. (2023) adopt a similar approach as ours, also accounting for measurement error in the PGIs, but focuses on sibling correlations rather than intergenerational associations in education. Rustichini et al. (2024) estimate empirical equations that bear some similarity with ours, yet their core focus is how cognitive and non-cognitive skills mediate the influence of the PGI on educational attainment, and they do not account for measurement error in the PGI. Finally, van Alten et al. (2025) estimate the causal effect of parental PGIs on children's outcomes, and how much of this effect can be explained by direct genetic transmission, using the Dutch Lifelines Biobank. Whilst complementary, their starting point is the effect of the parental PGI, not the intergenerational association as in our paper.

This paper is organized as follows. Section 2 introduces our theoretical framework, Section 3 describes the Data and Variables, Section 4 reviews the Methods, Section 5 presents the Results and Section 6 discusses and concludes.

2 Theoretical Framework

2.1 Conceptual framework

Our framework is a variation on the theoretical model of intergenerational mobility developed by Becker and Tomes (1979) and Solon (1999), and discussed in Holmlund et al. (2011). Our framework represents a two-generation model where offspring educational attainment Y_c depends on the child's genetic endowments G_c, parental educational attainments, Y_m and Y_f, environmental characteristics determined by parents, E_m and E_f[6], and child specific characteristics, e_c, which are orthogonal to the other regressors.

$$Y_c = \alpha + \rho G_c + \beta_m Y_m + \beta_f Y_f + \omega_m E_m + \omega_f E_f + e_c \tag{1}$$

The standardized genetic endowments of the child G_c are determined by the standardized genetic endowments of the parents, G_m and G_f, as well as by random *de novo* mutations, captured by ε_c.

$$G_c = \kappa_m G_m + \kappa_f G_f + \varepsilon_c \tag{2}$$

The theoretical expectation for κ_m and κ_f is 0.5 as parents each transmit half of their genetic material to their children.

Parental educational attainment Y_p depends on parental genetic endowments G_p, parental circumstances

[6]For notational simplicity, we assume that E and C below are variables but in practice they could be vectors of circumstances.

C_p, and on parent specific characteristics e_p that are orthogonal to the other regressors. Similarly, environments shaped by parents E_p depend on parental genetic endowments G_p, on parental circumstances C_p, and on parental specific characteristics η_p, which are orthogonal to the other regressors.[7]

$$Y_p = \alpha_{yp} + \rho_{yp} G_p + \lambda_{yp} C_p + e_p \quad \text{with } p = m, f \tag{3}$$

$$E_p = \alpha_{ep} + \rho_{ep} G_p + \lambda_{ep} C_p + \eta_p \quad \text{with } p = m, f \tag{4}$$

Figure 1 depicts the relationships graphically. The double arrowed dotted line represents assortative mating at the Educational Attainment (EA) level. As a byproduct of assortative mating at the level of parental education, there is an induced correlation across parents at the genotype level and in their (unobserved) circumstances.

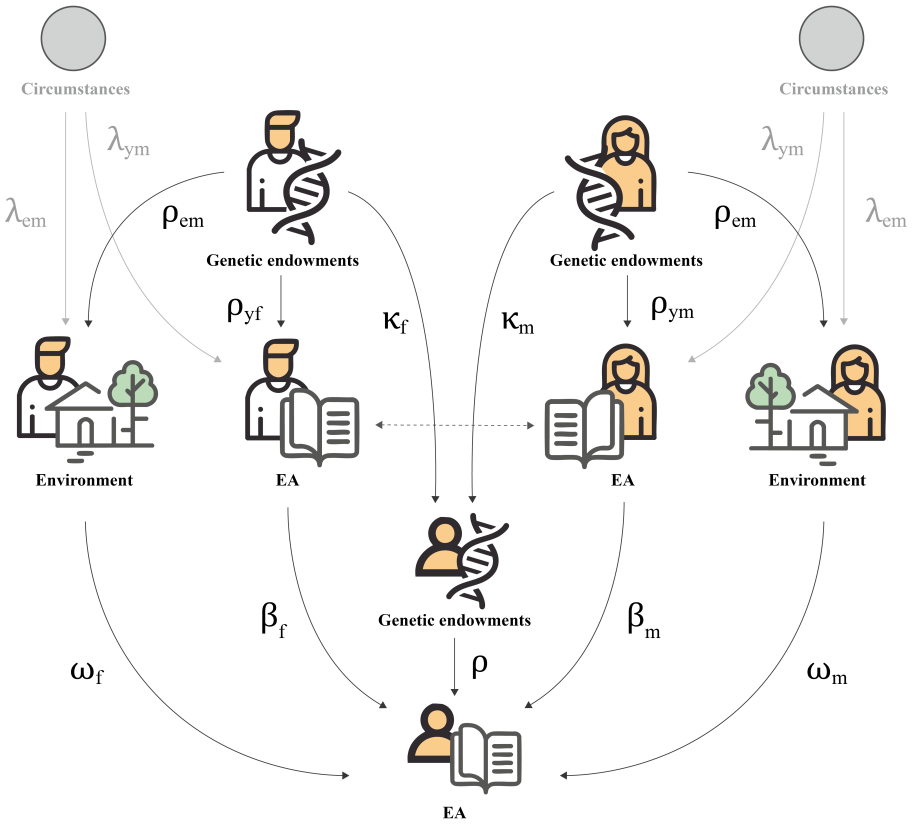

Figure 1 Directed Acyclic Graph (DAG) representing the causal paths between our interest variables.

[7] In practice, parental education may also influence the child's outcome through the childhood environment, but this is captured in the coefficient $\beta_p, p = m, f$.

In our baseline model, we adopt two assumptions:

A1. Two-generation model

$$\text{Cov}(G_p, C_p) = 0, \quad p = m, f$$

A2. Assortative mating is only at educational attainment level

$$\text{Cov}(E_f, Y_m | Y_f) = 0;$$

$$\text{Cov}(G_f, Y_m | Y_f) = 0 \quad \text{and vice-versa}$$

Assumption A1 implies that circumstances C_p are purely environmental. Environmental circumstances C_p can include variables such as income or education of grandparents, schools that parents attended, or neighborhoods where parents lived.

Assumption A2 states that assortative mating only exists at the educational attainment level. This implies that father's environmental circumstances E_f and genotype G_f are not correlated to the educational attainment of the mother, conditional on father's educational attainment, and vice versa.

We make use of these assumptions to render the derivations more transparent and to highlight the intuition of where differences between approaches arise. In practice, we expect grandparental genetic endowments to affect parent's genetic endowments and parental circumstances. Likewise, parents may sort on characteristics beyond educational attainment. Appendix F relaxes both assumptions, and the associated results are discussed in Section 5.3.

2.2 Broad and narrow genetic confounding

Consider the data generating process in equations (1) to (4). This section derives two types of genetic confounding: broad and narrow. As a baseline, consider a simple intergenerational association model in which only the maternal and paternal education are included

$$Y_c = \alpha + \beta_m Y_m + \beta_f Y_f + \varepsilon_c \tag{5}$$

The resulting Ordinary Least Squares (OLS) estimator for the coefficient of maternal educational attainment in this model follows from standard OLS logic (see Appendix A for the derivation):

$$\hat{\beta}_m = \beta_m + \underbrace{\underbrace{\rho\,\kappa_m \frac{Cov(G_m, \tilde{Y}_m)}{V(\tilde{Y}_m)}}_{\text{Direct genetic transmission}} + \underbrace{\omega_m \rho_{em} \frac{Cov(G_m, \tilde{Y}_m)}{V(\tilde{Y}_m)}}_{\text{Genetic nurture}}}_{\text{Broad genetic confounding}} + \underbrace{\omega_m \lambda_{em} \frac{Cov(C_m, \tilde{Y}_m)}{V(\tilde{Y}_m)}}_{\text{Environmental confounding}} \tag{6}$$

with $\tilde{Y}_m$ representing the residual of maternal education after conditioning on paternal education.

Hence, a naive intergenerational association between maternal and offspring educational outcome – conditional on paternal education – reflects the causal effect of maternal education β_m, but is confounded by direct genetic transmission (second term on the RHS), genetic nurture (third term on the RHS), and environmental confounding (fourth term on the RHS).

Our definition of narrow genetic confounding is equal to the term 'direct genetic transmission'. Direct genetic transmission is the bias caused by the transmission of parental genes to their offspring. Broad genetic confounding is defined as the sum of direct genetic transmission and genetic nurture. Given that we are considering a two-generation model, genetic nurture only represents the influence of parental genes on the rearing environment of their offspring (e.g., Kong et al., 2018, see Section F.2 of Appendix F for an extension).

2.3 Estimand recovered from molecular genetic data

In our empirical analysis, we are interested in using molecular genetic data to quantify what share of the intergenerational association from equation (6) is due to genetic confounding. The three coefficients of narrow genetic confounding $\rho\kappa_m\frac{Cov(G_m,\tilde{Y}_m)}{V(\tilde{Y}_m)}$ can be directly estimated from the data under our Assumptions 1 and 2.

To estimate broad genetic confounding, we add parental genetic endowments as control variables to the baseline regression (5).

$$Y_c = \alpha' + \beta'_m Y_m + \beta'_f Y_f + \phi_m G_m + \phi_f G_f + \varepsilon_c \tag{7}$$

The inclusion of parental genetic endowments will strip the original association from familial genetic confounding that acts through two channels: the effect of parental genes on the genes of the child (genetic transmission), and the effect of parental genes on the environment (genetic nurture):

$$\begin{aligned}
\hat{\beta}'_m &= \frac{Cov(Y_c,\tilde{Y}_m^{gc})}{V(\tilde{Y}_m^{gc})} \\
&= \frac{Cov(\rho G_c + \beta_m Y_m + \beta_f Y_f + \omega_m E_m + \omega_f E_f, \tilde{Y}_m^{gc})}{V(\tilde{Y}_m^{gc})} \\
&= \beta_m + \underbrace{\rho\frac{Cov(G_c,\tilde{Y}_m^{gc})}{V(\tilde{Y}_m^{gc})}}_{=0 \text{ (due to Mendel's Law)}} + \underbrace{\beta_f\frac{Cov(Y_f,\tilde{Y}_m^{gc})}{V(\tilde{Y}_m^{gc})}}_{=0 \text{ (due to conditioning)}} + \omega_m\frac{Cov(E_m,\tilde{Y}_m^{gc})}{V(\tilde{Y}_m^{gc})} + \underbrace{\omega_f\frac{Cov(E_f,\tilde{Y}_m^{gc})}{V(\tilde{Y}_m^{gc})}}_{=0 \text{ (due to A2)}} \\
&= \beta_m + \omega_m\lambda_{em}\frac{Cov(C_m,\tilde{Y}_m^{gc})}{V(\tilde{Y}_m^{gc})}
\end{aligned} \tag{8}$$

where $\tilde{Y}_m^{gc}$ represents the residual of Y_m conditional on Y_f, G_m and G_f.

As can be seen from equation (8), the coefficient estimate $\hat{\beta}'_m$ from equation (7) is not confounded by genetic transmission nor genetic nurture. It does not provide an unbiased estimate of the causal effect of maternal education on the child's education β_m either, because the estimator is confounded by maternal environmental circumstances C_m. Still, controlling for parental genotypes strips the association from broad genetic confound-

ing. As a result, comparing (8) with (6) shows that an estimate for broad genetic confounding is given by $\hat{\beta}_m - \hat{\beta}'_m$ (see equation (14) in Appendix A).[8]

2.4 Comparison to other approaches

In this section we describe how our approach compares to existing approaches in the literature: controlling for children's genetic endowments, adoption studies, egg or sperm donor studies, and children-of-twins studies. A full derivation including graphical explanations is provided in Appendix B.

Controlling for the child's genetic endowments, as implemented in Isungset et al. (2022), aims to estimate narrow genetic confounding. However, since children's genes correlate approximately 50% with their parents' genes (see Appendix B.1), the method additionally includes a share, but not all, of genetic nurture. Consequently, estimates derived from this approach are hard to interpret, as they lie in between narrow and broad genetic confounding.

Adoption studies (e.g., Sacerdote, 2011; Holmlund et al., 2011, see Appendix B.2) compare the intergenerational association of adopted with non-adopted children. They aim to estimate narrow genetic confounding, by breaking the direct genetic transmission channel. Similar to our baseline method, they rely on Assumption 2 and a variation of Assumption 1 – that adoptees are randomly assigned to families. This ensures no correlation between the genetic endowments of adoptive parents and children, and between characteristics of biological and adoptive parents. However, since biological parents provide their genes and influence in-utero and early childhood environments, adoption studies capture a combination of narrow genetic confounding as well as early-life environmental differences.

Studies using donor-conceived children (Rasmussen et al., 2024, see Appendix B.3) are conceptually similar to adoption studies, as they break the genetic transmission link. Narrow genetic confounding is estimated by comparing coefficients between donor-conceived and biological children. This approach has the advantage that donors are not expected to influence in-utero or early childhood environments. A limitation is that donor designs usually only break the genetic transmission link for one parent, such that their estimates capture a share of the partner's genetic and non-genetic characteristics through assortative mating. Further, and similar to adoption designs, parents who resort to IVF might be different that those who do not, potentially conflating the comparison between donor-conceived and biological children (see Equation 20).

Children-of-twins studies compare offspring of identical twins relying on the notion that identical twins

[8] Whereas $\hat{\beta}_m - \hat{\beta}'_m$ does not include genetic confounding, there is a modest bias in the estimation of broad genetic confounding arising from estimating the influence of environmental characteristics conditional on parental genotypes in (8):

$$\omega_m \lambda_{em} \left[\frac{Cov(C_m, \tilde{Y}_m)}{V(\tilde{Y}_m)} - \frac{Cov(C_m, \tilde{Y}_m^{gc})}{V(\tilde{Y}_m^{gc})} \right] \tag{9}$$

The term within brackets is equal to the difference between the regression coefficient of Y_m on environmental circumstances C_m in two separate regressions. In the first regression, Y_m has been residualized with respect to Y_f, G_m and G_f. In the second regression, Y_m has been residualized with respect to Y_f alone. In Table F3 in Appendix F.3 we show for a list of environmental variables that the term within brackets is very small, and therefore can be seen as a rounding error in the estimation of broad genetic confounding.

share 100% of their genetic endowments (see Appendix B.4). By holding constant one of the parents genetic endowments, they aim to estimate broad genetic confounding. Theoretically speaking, this method is identical to our estimate of broad genetic confounding, with two exceptions. First, it typically breaks the link for only one parent, since it is very rare that both parents of a child come from a monozygotic twin pair. Second, twin samples might not be comparable to non-twin samples. In particular, given the likelihood of geographical and social proximity among twins, it is possible that aunts and uncles maintain closer relationships—and thus exert stronger genetic nurture effects—on their nieces and nephews.

The theoretical framework clarifies which type of confounders are being captured by each methodology. In particular, it shows how adoption and children-of-twins designs capture different sources of genetic confounding, and therefore cannot be compared directly. It further sheds light on key assumptions and limitations of each method. Under Assumptions 1 and 2 (or variations), using molecular genetic data has a few key advantages. It can account for genetic influences from both parents, and, unlike the adoptee or donor-conception designs, do not rely on comparisons between potentially different samples. A second advantage is that, with appropriate data, using molecular genetic data enables the estimation of both narrow and broad genetic confounding within the same sample. A third and final advantage is the arguably improved external validity of results derived from this method.

3 Data

This section introduces our dataset, defines our main variables and introduces summary statistics. The Avon Longitudinal Study of Parents and Children (ALSPAC) is a prospective and longitudinal study of children and parents (Boyd et al., 2013; Fraser et al., 2013). The data collection aimed to track children from fetal life, through infancy into adolescence and young adulthood. 20,248 Pregnant women resident in Avon, United Kingdom, with an expected delivery date between April 1, 1991, and December 31, 1992, were identified as being eligible and invited to take part in ALSPAC. 14,541 eligible pregnant women were enrolled at baseline. From these pregnancies, 13,988 children were alive at one year of age.[9] More details about this study can be found in Fraser et al. (2013).[10]

Most children and mothers were genotyped, and a subsection of 1,283 trios – mothers, fathers and chil-

[9] When the oldest children were approximately 7 years of age, an attempt was made to bolster the initial sample with eligible cases who had failed to join the study originally. As a result, when considering variables collected from the age of seven onward (and potentially abstracted from obstetric notes) there are data available for more than the 14,541 pregnancies mentioned above: The number of new pregnancies not in the initial sample (known as Phase I enrolment) that are currently represented in the released data and reflecting enrollment status at the age of 24 is 906, resulting in an additional 913 children being enrolled (456, 262 and 195 recruited during Phases II, III and IV respectively). The phases of enrollment are described in more detail in Fraser et al. (2013); Northstone et al. (2019). The total sample size for analyses using any data collected after the age of seven is therefore 15,447 pregnancies, resulting in 15,658 foetuses. Of these 14,901 children were alive at 1 year of age.

[10] ALSPAC's study website contains details of all the available data through a fully searchable data dictionary and variable search tool, see http://www.bristol.ac.uk/alspac/researchers/our-data/.

dren[11]– were genotyped. We exploit the existence of mother-child duo's to impute the paternal genomes of an additional 4,230 fathers based on the method proposed by Young et al. (2022, details in Appendix D). By using this method, our sample of trios is expanded to 5,513 trios. Our final baseline sample consists of 4,032 mother-child-father trios with information about the outcome (Key Stage 4) and main explanatory variables (maternal and paternal years of education, and educational attainment polygenic indices, or PGIs).

3.1 Variables

Outcome variable

Offspring Key Stage 4: The Key Stage 4 refers to the total point score the students obtained in the General Certificate of Secondary Education (GCSE) (or equivalent) examinations, taken at age 16. The total point score aggregates the scores of eight GCSEs exams including English, mathematics, sciences (physics, chemistry, biology, computer science), history, geography, and an ancient or modern foreign language. The Key Stage 4 examination is the last occasion in the educational system where pupils are assessed along with all their peers, as it falls within the compulsory schooling for ALSPAC pupils. This means that while earlier Key Stages exist, the Key Stage 4 is the highest stakes examination that is mandatory for all pupils, and therefore it comprises our main outcome of interest.[12] This variable is retrieved from administrative records (National Pupil Database), a census of all pupils in England within the state school system, which is matched to ALSPAC.[13] The final score ranges from 0 to 1171, with a mean of 437 and standard deviation 134 (see Table 1).

Explanatory variables

Mother and father years of education: Maternal and partner's highest educational attainment was self-reported by the mother at 32 weeks of gestation. The father's education level was included only if the mother identified her current partner as the unborn child's father at 8 weeks of pregnancy. Maternal highest educational attainment ranges from having no education (0.02%), having a CSE (certificate of secondary education, 10.12%), vocational education (9.05%), O-levels (38.62%), A-levels (27.03%), to having a degree (15.15%). The father's highest education qualification varies from having a CSE (certificate of secondary education, 16.25%), vocational education (9.00%), O-levels (24.83%), A-levels (30.23%) to having a degree (19.69%). The conversion into years of education is based on van den Berg et al. (2022) and assigns 0 years of education for no

[11]Of the original 14,541 initial pregnancies, 338 were from a woman who had already enrolled with a previous pregnancy, meaning 14,203 unique mothers were initially enrolled in the study. As a result of the additional phases of recruitment, a further 630 women who did not enrol originally have provided data since their child was 7 years of age. This provides a total of 14,833 unique women (G0 mothers) enrolled in ALSPAC as of September 2021.G0 partners were invited to complete questionnaires by the mothers at the start of the study and they were not formally enrolled at that time. 12,113 G0 partners have been in contact with the study by providing data and/or formally enrolling when this started in 2010. 3,807 G0 partners are currently enrolled.

[12]Another obvious outcome to study intergenerational persistence of educational outcomes is completed education. In our data, we only have self-reported educational attainment for 241 children at the age 23+, such that large attrition makes this a difficult outcome to study.

[13]At age 18, study children were sent 'fair processing' materials describing ALSPAC's intended use of their administrative records and were given clear means to consent or object via a written form. Data were not extracted for participants who objected, or who were not sent fair processing materials.

education qualification, 11 years of education to CSE or O-level education, 12 years to vocational education, 13 years to A-levels and 16 years to a college or university degree.

	Mean	S.D.	Min.	Max.	*N*
Key stage 4	436.91	133.59	0	1171	4,032
Mother years of education	12.39	1.75	0	16	4,032
Father years of education	12.68	1.84	11	16	4,032
EA PGI child (23andMe)	0.00	1.00	-4	3	4,032
EA PGI child (UKB)	0.00	1.00	-3	3	4,032
EA PGI mother (23andMe)	0.00	1.00	-4	4	4,032
EA PGI mother (UKB)	0.00	1.00	-3	4	4,032
EA PGI father (23andMe)	0.00	1.00	-3	4	4,032
EA PGI father (UKB)	0.00	1.00	-2	4	4,032

Table 1 Summary statistics of the baseline sample (ALSPAC). S.D.=Standard deviation; Min.=Minimum; Max.=Maximum; PGI=Polygenic index

Control variables

We control for genetic confounding with the parental Polygenic Indices (PGIs) (see section 3.2 below). Since parental education is measured during pregnancy, virtually all other control variables are potentially endogenous ("bad") control variables. Our baseline estimates therefore do not include any control variables and we start with the raw association between parental and child's educational outcomes. In robustness checks, we include gender and the first 20 principal components of the genetic relatedness matrix of the mother and child, and the results are virtually identical (see Appendix G.2).

3.2 The Educational Attainment Polygenic Index (EA PGI)

Construction and predictive power of the EA PGI Humans share more than 99 percent of their DNA, yet there are variations at specific loci within the genome. The most common type of genetic variation across humans are so-called single nucleotide polymorphisms (SNPs). At each SNP location there can be two different variants (or alleles). It is common to measure SNPs by counting the number of minor alleles (the variant that occurs least frequently in the population). Since a child inherits two copies of each genetic variant, one from each parent, a SNP can take the values 0, 1 or 2. A PGI is constructed by adding up individual SNPs, where each SNP is weighted by the strength of the association between the SNP and the outcome variables as estimated in a Genome-Wide Association Study (GWAS) (Dudbridge, 2013). A PGI therefore exploits the joint predictive power of multiple SNPs for a particular outcome.

The predictive power of PGIs increases with the sample size of the underlying GWAS (Dudbridge, 2013). To avoid overfitting in PGI analyses, the discovery sample – the sample used to obtain the SNP weights – should be independent from the baseline sample. In our study, we used the summary statistics based on the UK Biobank and the 23andMe data set, constructed by Muslimova et al. (2025), such that we could construct two

independent PGIs for each individual. The weights were corrected for linkage disequilibrium (LD; structural correlation across SNPs in the genome) using the software LDpred (Vilhjálmsson et al., 2015). The resulting PGIs were standardized such that the mean is zero and the standard deviation is 1 for our baseline sample. Figure 2 graphically shows the predictive power of the child's EA PGIs on the outcome, KS4, reaching an R-squared of roughly 7-8%. Appendix C offers a more comprehensive primer on genetics, heritability and predictive power of PGIs. Details about the genotyping procedure, construction of the PGI, and imputation of paternal genotypes can be found in Appendix D.

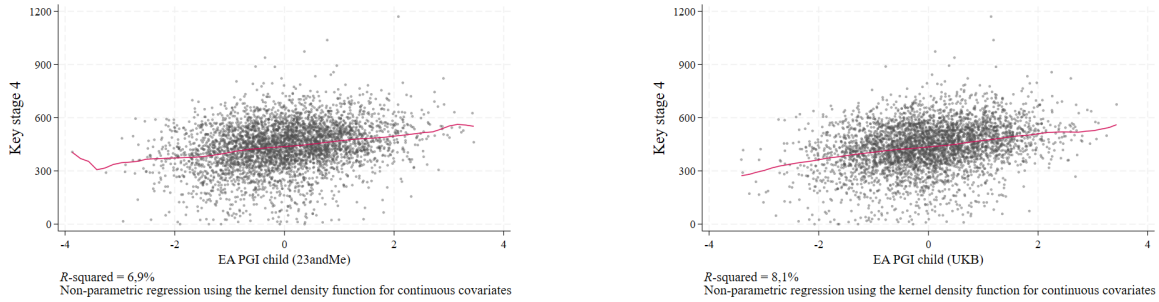

Figure 2 Scatterplot of Key Stage 4 and the EA PGI of the child with a locally weighted regression line. On the left, the PGI based on the summary statistics from the 23andMe data set and on the right, based on the summary statistics from the UKBiobank.

Interpretation of the EA PGI: The EA PGI aggregates several genetic variants that predict years of education (Rietveld et al., 2013; Okbay et al., 2016; Lee et al., 2018; Okbay et al., 2022). In doing so, the EA PGI reflects the best linear genetic predictor of education (Mills et al., 2020). Besides predicting general cognitive ability, it also predicts personality traits (Openness, Conscientiousness, Agreeableness) (Smith-Woolley et al., 2019).

While the EA PGI is a promising proxy for genetic-based advantage in Western educational systems, its interpretation calls for a few words of caution. In particular, the predictive power of the EA PGI is dependent on societal structures. Indeed, we observe different associations between a PGI and a certain trait depending on birth year (Papageorge and Thom, 2020; Lin, 2020; Herd et al., 2019), socio-economic status, age, or sex (Mostafavi et al., 2020). This means that the links between genetic variants and traits are likely mediated by the environment, such that the same genetic variants might have a different effect on a given trait depending on the environmental context (e.g., Visscher et al., 2017). For the remainder of this paper, the EA PGI is interpreted as having a genetic based advantage to succeed in Western schooling system – but this genetic advantage is not a biological, immutable law, but rather environmentally and contextually dependent.

Indirect effects, confounding, and measurement error Since a PGI is typically constructed based on a GWAS that did not control for parental SNPs, a PGI reflects both direct and indirect genetic effects (Biroli et al., 2025). Direct effects refer to the causal impact of an individual's own genes on their outcome. Indirect

effects arise from two sources: genetic nurture and population stratification. Genetic nurture refers to the influence of relatives' genes on an individual's outcome. In our definition, genetic nurture is included in broad genetic confounding. Population stratification arises from differences in genetic structure and allele frequencies across subgroups or ethnicities, and can induce spurious associations if these subgroups additionally vary in the outcome because of non-genetic reasons (Young et al., 2019). Population stratification is not part of broad genetic confounding. In our baseline model we restrict to two generations, and therefore only distinguish between direct genetic effects and genetic nurture, assuming no population stratification. We relax this assumption in section 5.3. In practice, since the ALSPAC sample is highly homogeneous from a small geographical area (Ruisch et al., 2019), population stratification is minimal.[14]

Current PGIs likely underestimate the true magnitude of genetic effects (e.g., Wray et al., 2019). One explanation is the fact that PGIs are context dependent such that the weights vary across samples and contexts (de Vlaming et al., 2017). We try to tackle this issue by analyzing different PGIs from different discovery samples, one of which is from the same context as our analysis sample (UK). Another proposed explanation is that PGIs only use common SNPs, such that rare variants are excluded (Young, 2019). A final source of measurement error is that the PGIs aggregate coefficients estimated on a finite discovery sample, leading to classical measurement error (van Kippersluis et al., 2023). Here we tackle the issue of measurement error by using two PGIs constructed based on independent GWAS samples (DiPrete et al., 2018; van Kippersluis et al., 2023, see section 4 for details).

4 Methods

Our goal is to quantify how much of the association between parental educational attainment (EA) and the child's KS4 outcome is biased by genetic confounding. We start by estimating the partial correlation between the Key Stage 4 outcome and parental educational attainment measured in years, for both mothers (EA_m) and fathers (EA_f). This is depicted in Equation (10).

$$KS4 = \alpha^0 + \beta_m^0 EA_m + \beta_f^0 EA_f + \varepsilon^0 \tag{10}$$

To account for assortative mating, we include the educational attainment of both parents simultaneously in our model, allowing us to estimate the effect of each parent's education net of the other's influence (Holmlund et al., 2011). We also study mother's and father's educational attainment separately (see Appendix G.8).

Next, we control for the parents' EA PGI – PGI_m and PGI_f – in Equation (11).[15] Under the model assump-

[14]The standard approach to adjust for population stratification is to include principal components from the genetic relatedness matrix. Controlling for principal components does not affect our results (see Appendix G.2), yet we cannot fully rule out the presence of more subtle forms of population stratification (see Appendix F).

[15]Adding the child's PGI as control should not further alter the estimates of β_m^1 and β_f^1 as conditional on parental PGI's, the child PGI is random and therefore should be uncorrelated with any predetermined parental characteristics, including parental education. Appendix

tions from Section 2 and if the parental PGIs are accurate measures of G_f and G_m, controlling for parental PGIs clears β_m^1 and β_f^1 of genetic confounding by direct genetic transmission as well as genetic nurture.

$$KS4 = \alpha^1 + \beta_m^1 EA_m + \beta_f^1 EA_f + \delta_m^1 PGI_m + \delta_f^1 PGI_f + \varepsilon^1 \tag{11}$$

To overcome measurement error of the PGIs, we construct two independent PGIs for the same trait and perform a Two-Stage Least Squares (2SLS) regression, with one PGI instrumenting for the other (DiPrete et al., 2018). We implement this using Obviously Related Instrumental Variables (ORIV, Gillen et al., 2019; van Kippersluis et al., 2023), which makes the most efficient use of the information in the two independent PGIs and avoids having to arbitrarily select one PGI as IV for the other.[16] The coefficients β_m^1 and β_f^1 are compared with the coefficients β_m^0 and β_f^0 to calculate the fraction of intergenerational transmission explained by broad genetic confounding.

To estimate narrow genetic confounding (see Section 2), we compute $\rho\,\kappa_m \frac{Cov(G_m,\tilde{Y}_m)}{V(\tilde{Y}_m)}$ for the mother, and vice versa for the father. We estimate $\hat{\rho}$ as the coefficient of the child's EA PGI in a regression with the child's KS4 score as dependent variable and the parental EA PGIs as controls. We set $\kappa_m = 0.5$ as children inherit 50% of each their genetic variants from each parent. Finally, we estimate $\frac{Cov(G_m,\tilde{Y}_m)}{V(\tilde{Y}_m)}$ as the regression coefficient of Y_m on G_m, where Y_m has been residualized with respect to Y_f. In all regressions, we account for measurement error and standardization of the PGIs (see Appendix E for detail).

5 Results

5.1 Intergenerational association and genetic confounding

Column 1 of Table 2 shows the results of Equation (10), the simple intergenerational association between offspring test scores and parental years of education. The coefficients β_m^0 and β_f^0 are 14.2 and 12.3, respectively. Thus, an additional year of maternal and paternal education is associated with scoring 14.2 and 12.3 points higher on the Key Stage 4 test (or roughly 3% relative to a mean of 436.91), respectively. Column 2 shows that every standard deviation increase in the maternal EA PGI is associated with a 25.1 points higher KS4 test score (or roughly 5.7%), and that every standard deviation increase in the paternal EA PGI is associated with a 25.6 points higher KS4 test score (or roughly 5.9%).

Column 3 shows the results of Equation (11), i.e., the intergenerational association with controls for the parents' EA PGIs. The coefficients β_m^1 and β_f^1 decrease to 10.5 and 9.6, showing that controlling for the

G.10 empirically verifies this conditional independence.

[16] Using ORIV, we are able to boost the implied variance explained of the child EA PGI from 8.1% to 27.8% (see Table G23). This shows that the correction for measurement error is consequential. The estimates are in the same ballpark as SNP-based heritability estimates for the individual GCSE items of 15-22% reported in Rimfeld et al. (2015) and 31% reported in Krapohl and Plomin (2016), suggesting that we are successful in correcting for classical measurement error.

parental EA PGIs strips the original association of some of the genetic confounding effect. The reduction is approximately 26% and 22% for the mother's and father's years of education, respectively.

Column 4 regresses the KS4 results on parental and child's EA PGI to obtain the causal effect of the child's EA PGI on their test scores - ρ. The results show that an additional standard deviation of the EA PGI of the child increases test scores by 21.3 points (roughly 4.9%).

Columns 5, 6, and 7 replicate columns 2, 3, and 4, but using ORIV, where each PGI is instrumented with the other independent PGI for educational attainment to correct for measurement error.[17] Using this method, the coefficient of the parental EA PGIs increases, from 25.1 and 25.6 in column 2, to 30.2 (7.0% relative to a mean of 436.91) and 32.7 (7.5%) in column 5. The coefficient of the child's EA PGI also increases, from 21.3 in column 4 to 39.8 (9.1%) in column 7. This implies that measurement error in the EA PGI is non-negligible. Finally, applying ORIV to parental PGIs further reduces the coefficients β_m^1 and β_f^1 estimated in column 3, from 10.5 and 9.6 to 7.8 and 7.2 in column 6, respectively.

	OLS				ORIV		
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Mother years of education	14.214***		10.460***			7.797***	
	(1.305)		(1.354)			(1.072)	
Father years of education	12.334***		9.568***			7.224***	
	(1.246)		(1.249)			(0.949)	
EA PGI mother		25.094***	14.543***	14.737***	30.166***	20.678***	10.678***
		(1.978)	(2.074)	(2.861)	(2.039)	(2.284)	(3.182)
EA PGI father		25.585***	18.765***	10.988**	32.687***	26.397***	5.516
		(1.980)	(2.006)	(3.516)	(2.104)	(2.176)	(4.067)
EA PGI child				21.316***			39.771***
				(4.241)			(5.049)
Broad genetic conf. mother			3.754			6.417	
(in %)			(26.4%)			(45.1%)	
Broad genetic conf. father			2.765			5.109	
(in %)			(22.4%)			(41.4%)	
Narrow genetic conf. mother				1.896			4.881
(in %)				(13.3%)			(34.3%)
Narrow genetic conf. father				1.103			2.929
(in %)				(8.9%)			(23.7%)
R-squared	0.097	0.082	0.127	0.087	-	-	-
N	4,032	4,032	4,032	4,032	4,032	4,032	4,032

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Table 2 Results of the OLS and ORIV regressions explaining the offspring Key Stage 4 grade. Robust standard errors in parentheses.

Using OLS based on a single PGI (column 3) suggests that broad genetic confounding is responsible for 3.7

[17] Appendix G.5 shows the first stage regressions. The independent PGIs are highly predictive of each other and the F-statistic varies between 400 and 1500 (see Table G13). Appendix G.5 also repeats the results using two PGIs based on summary statistics of two independent halves of the UK Biobank data set. Arguably, the assumptions of (OR)IV are more likely to hold when the PGIs are constructed from the exact same discovery sample rather than two discovery samples from different countries. The results are similar.

and 2.7 of the original association for mothers and fathers, respectively. Column 7 shows that ORIV produces much larger values, of 6.4 and 5.1, respectively. Using ORIV and taking the ratio of our estimated broad genetic confounding with respect to the original association in column (1), we estimate that broad genetic confounding explains 45.1% and 41.1% of the parent-child education association for mothers and fathers, respectively.

Similarly, using OLS based on a single PGI suggests that narrow genetic confounding is 2.7 and 2.1, while using ORIV reveals much larger values, 4.7 and 4.0, for mothers and fathers, respectively.[18] Again comparing to column (1), narrow genetic confounding explains 33.7% and 32.4% for mothers and fathers, respectively, or around 75% of broad genetic confounding.

5.2 Robustness and heterogeneity

In this section, we probe the robustness of our baseline specification by introducing alternative model specifications. While in our main specification we use the total points in the Key Stage 4 exam as our main outcome variable, in Appendix G.1 we use the average points per Key Stage 4 examination, and the highest points obtained in the Key Stage 4 Science examination instead. The results are very similar in both cases. In Appendix G.2 we include gender and a set of principal components of the genetic relationship matrix as basic controls. Table G9 shows that the results are virtually identical. In Appendix G.3 we present estimates separately for boys and girls. The results are very similar across genders.

Appendix G.4 presents the results when parental education is measured in levels. Higher educational levels are always associated with larger KS4 results, with the raw associations monotonically increasing in paternal education and close to linear. We observe a proportional reduction of around 30-40% in the coefficients of educational levels when controlling for parental PGIs, which varies a little depending on the reference category and seems slightly higher at higher levels of education.

Our main ORIV estimates rely on instrumenting the EA PGI based on UK Biobank summary statistics with the EA PGI based on 23andMe summary statistics, and vice versa. Appendix G.5 presents the results for the individual IV estimates, as well as ORIV estimates where we split the UK Biobank discovery sample into two equal halves. Arguably, using two UK Biobank-based EA PGIs as instruments for each other is more likely to satisfy the assumption that the genetic correlation between the two independent PGIs is 1 (DiPrete et al., 2018). Broad genetic confounding is slightly lower in this specification, estimated at 35-36%, likely due to the smaller predictive power of the PGIs resulting from smaller discovery samples.

Although imputing the father's genotype from the child's and mother's information enhances statistical power by increasing sample size and leads to unbiased estimates (Young et al., 2022), the imputation remains an approximation rather than an exact representation. In Appendix G.6 we present our results for the non-imputed

[18] Narrow genetic confounding is estimated as $0.5\hat{\rho}\frac{Cov(G_p,\tilde{Y}_p)}{V(\tilde{Y}_p)}$. $\hat{\rho}$ is estimated in columns (4) and (7). Using OLS, the final term is estimated as 0.178 and 0.104 for mothers and fathers, respectively, while using ORIV the terms are 0.245 and 0.147 for mothers and fathers, respectively. See Appendix E for details.

sample of 1,022 trios with observed genetic information. Not surprisingly, standard errors increase as a result of the smaller sample size. More substantially, we observe that broad genetic confounding is estimated to be somewhat smaller than in our baseline specification (24.2-31.4%), with larger differences between mothers and fathers. Part of this discrepancy arises from the differences in the baseline sample, as the simple associations also differ in comparison to our baseline estimates.

It is possible that intergenerational persistence also arises from a non-additive interplay between or within genetic and environmental factors. Table G20 in Appendix G.7 presents the results with interaction terms. We find the interactions between mother's and father's education to be small and insignificant. We do detect a statistically significant interaction between the maternal and paternal PGIs, with the returns to one parent's PGI declining as the partner's PGI increases. Accounting for this interaction however leads to similar estimates of broad genetic confounding. Finally, we find the interactions between the PGI's and maternal and paternal education to be statistically insignificant and quantitatively small. Clearly, the endogeneity of parental education as well as the additive construction of the EA PGI may prevent us from detecting subtle forms of $G \times E$ (Biroli et al., 2025). However, our results are at least suggestive that while both genetic and environmental transmission are quantitatively important, the interaction between genetic and environmental factors appears quantitatively less important (see also Isungset et al., 2022).

Finally, we study the influence of maternal and paternal years of education separately in Appendix G.8. As anticipated, the raw associations between a single parent's years of education and the child's KS4 score are higher when spousal education is not controlled for. This occurs because the association partially reflects the influence of spousal educational attainment through assortative mating. The role of broad genetic confounding in this specification is around 30%, a little smaller than our baseline scenario where we include both parents simultaneously. This specification contains a bias term as assortative mating is stronger at the educational level (correlation of 0.52) than at the genome level (correlation of 0.13, not shown). Consequently, the simple association captures a larger share of the partner's influence, while the subsequent regression controlling for a single parental PGI removes a smaller proportion of the partner's genetic confounding. As such, we find that is not innocuous to omit one of the parents.

5.3 Relaxing assumptions

Our theoretical model assumes (A1) a two generation model where parental circumstances are environmental and uncorrelated with parental genetics, and (A2) assortative mating occurs only on education. These assumptions simplify the derivations, and transparently show where differences across approaches stem from. Appendix F explores the impact of relaxing these assumptions. We find that our estimates of both broad and narrow genetic confounding are slightly overestimated.

Appendix F.2 shows that our estimates of narrow genetic confounding may be subject to an upward bias.

To understand this intuitively, note that the term $\frac{Cov(G_p,\tilde{Y}_p)}{V(\tilde{Y}_p)}$ is equal to $\frac{\rho_{yp}}{V(\tilde{Y}_p)}$, where ρ_{yp} is the causal effect of parental G on parental education Y (see Equation 39). Under assumptions A1 and A2, the causal effect ρ_{yp} can be estimated in the full sample. Relaxing A1 reveals that this coefficient also reflects population stratification and genetic nurture —- for example, the influence of grandparents on parental education. Using a subsample of sibling mothers ($N = 195$) and applying sibling fixed effects, we estimate the causal effect of a mother's PGI on her years of education, ρ_{ym}. Plugging in this value indicates that narrow genetic confounding accounts for roughly 18% of the original mother–child association.

For broad genetic confounding, the primary threat to our estimates comes from assortative mating on traits other than education (A2; see Equation 42, third term). In simple terms, if assortative mating does not just take place at the level of educational attainment, then the father's genetic endowments may still correlate with mother's educational attainment even conditional on father's educational attainment. We gauge the size of the bias using two different approaches. First, we directly compute the association between father's PGI and mother years of education conditional on father's years of education. We demonstrate that conditioning on education captures the majority of assortative mating at the genomic level, but not all. Second, we include the partner's PGI in our baseline estimation – that is, the baseline regression for mothers is a regression of the child's test score on maternal years of education, father's years of education, as well as the father's PGI. This effectively strips the baseline estimation from conditional assortative mating (see Equations 44 and 45) and ensures that we are estimating broad genetic confounding without the influence of parental assortative mating. Both approaches give remarkably similar estimates, and result in an estimate of broad genetic confounding of around 32% for mothers and 29% for fathers.[19]

5.4 Instrumenting for parental education

Whereas the results in Table 2 provide a novel and compelling way to estimate genetic confounding in intergenerational persistence of educational outcomes, the method stops short of establishing a causal effect of parental education on offspring outcomes. This is because genetic confounding is not the only possible source of confounding, and the remaining association between parental education could still reflect other unobserved environmental correlates of parental education. In earlier work, Dickson et al. (2016) exploit the introduction of the 1972 RoSLA reform as an exogenous increase in parental educational attainment in ALSPAC to study its influence on offspring test scores. In Appendix H we replicate their results in the full sample. The reduced form results suggest that if mothers and fathers were induced by the RoSLA reform to stay an extra year in school, their offspring's KS4 test scores would increase by 15-25 points (or 3.5-6%). At face value, these results therefore suggest that a sizable share of the remaining environmental association can be attributed to a

[19]The remaining sources of bias—stemming from environmental confounding and population stratification—are residuals and scaled by factors that are very close to zero. Additionally, the genetic homogeneity of the ALSPAC sample substantially limits bias stemming from population stratification. See Appendix F.3 for details.

causal effect of parental education on offspring test scores.

There are, however, some caveats to this interpretation. First, as is well-known, IV estimates a local average treatment effect based on compliers – i.c., fathers and mothers who would have liked to drop out of school in the absence of the reform, but were forced to stay due to the new rules – whereas our intergenerational association seeks to capture an average treatment effect on the treated. It is therefore impossible to directly compare the effect sizes and make claims regarding the share of intergenerational association stemming from a causal effect of parental education. Second, whereas the reduced-form results of Dickson et al. (2016) are relatively stable, the first stage estimates suggest that the results may suffer from weak instrument bias according to recent standards (Lee et al., 2022), especially for fathers for whom the birth date was often imputed. The sample size of ALSPAC may just be too small with too few fathers and mothers around the cutoff of 1 September 1957. Indeed, in our reduced sample of parent-offspring trio's for whom we observe genetic data, we were not able to replicate their results. While the lack of an effect in our sample may partly be due to selection into the sample who were genotyped, it plausibly also derives simply from the sheer reduction in sample size that renders our first stage estimates to being borderline significant (mothers) or even of opposite sign (fathers).

Overall, our take-away from this analysis is that there is suggestive evidence from ALSPAC as well as other studies (see Mogstad and Torsvik, 2022, for a recent review) to believe that some part of the environmental association between parental education and offspring test scores stems from a causal effect of parental education. However, data limitations prevent us from decomposing the environmental component into a causal effect and unobserved correlates of parental educational attainment.

6 Discussion and conclusion

This paper exploits molecular genetic data on 4,032 mother-father-child trios to quantify the role of genetic confounding in the intergenerational transmission of educational attainment. We go beyond the literature by developing a theoretical framework that incorporates empirical findings from the social genetics literature – such as genetic nurture and population stratification – and by using two independent PGIs for educational attainment to reduce measurement error in the genetic measures. Our results reveal that direct genetic transmission, or narrow genetic confounding, accounts for 18-33% of the intergenerational persistence in education, while broad genetic confounding accounts for 30-45%. We find no meaningful differences between fathers and mothers.

How do our results compare and add to the literature? Holmlund et al. (2011) review the literature and conclude that twin studies indicate that mother's schooling has little impact, whereas father's schooling seems more important in explaining children's educational outcomes. In other words, broad genetic confounding is estimated to be quantitatively meaningful, and larger for mothers than for fathers. Adoptee studies find narrow genetic confounding estimates that ranges from 0-47% for fathers and 0-71% for mothers (Holmlund et al.,

2011), with if anything again larger effects of father's education than mother's education. Interestingly, and completely opposite to this conclusion, Rasmussen et al. (2024) uses children conceived using sperm or egg donors to find an estimate of narrow genetic confounding of 100% for fathers and 0% for mothers, at least for reading outcomes. In sum, estimates vary widely, although one recurring finding is sizeable differences in the magnitude of maternal and paternal genetic confounding (Holmlund et al., 2011).

In contrast, our study aligns closely with the results found using molecular genetic data by Isungset et al. (2022), even though they are based on different samples and countries – United Kingdom vs Norway. While we find OLS broad genetic confounding estimates of 23-26% before correcting for measurement error, Isungset et al. (2022) finds OLS broad genetic confounding estimates of 15-18%. Similar to theirs, and unlike other methodologies, we also find very similar estimates for mothers and fathers. Whereas our framework demonstrates theoretically why different methodologies might differ, an important area of future research is to empirically reconcile these differences across molecular genetic versus twin, adoptions and donor-conception studies, preferably in the same population.

This study has a few limitations. While we implement solutions to deal with the measurement error in PGIs, these solutions might not be perfect. PGIs only capture additive effects of common genetic variants and not gene-gene interactions or rare genetic variants. Whereas the variance explained by rare coding variants is much smaller than the one explained by PGIs (Chen et al., 2023), we cannot rule out some underestimation of the importance of genetic confounding. Another limitation is that PGIs might capture subtle forms of population stratification, which can't be fully controlled for by employing principal components. A final limitation concerns the fact that individuals that choose to supply their DNA can be different than the ones who do not (e.g., Domingue et al., 2017; van Alten et al., 2024). Finally, parental education is self-reported and therefore may be subject to measurement error. We expect this issue to be minor for mothers, since years of education are derived from the highest degree attained, which is a salient and easy to remember life achievement. By contrast, reports of the partner's education may involve a larger degree of measurement error.

Despite those limitations, using molecular genetic data in economics research is a promising avenue that has the potential to advance our understanding of inequality and its persistence across generations. Using molecular genetic data to inform the field of intergenerational education persistence improves our theoretical understanding of genetic transmission and serves as an important test of previous methods. By employing direct genetic measures, we can directly test some assumptions of our theoretical framework, and differentiate between direct genetic transmission (narrow genetic confounding) and genetic nurture (part of broad genetic confounding) in the same sample. This uncovers novel and more subtle aspects of the broader role of the family in human capital production (Heckman and Mosso, 2014).

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A Theoretical framework derivations

Under the true data generating process specified in equations (1) to (4), and when estimating equation (5), the resulting estimate (equation 6 in the main text) is given by:

$$
\begin{aligned}
\hat{\beta}_m &= \frac{Cov(Y_c, \tilde{Y}_m)}{V(\tilde{Y}_m)} = \frac{Cov(\rho G_c + \beta_m Y_m + \beta_f Y_f + \omega_m E_m + \omega_f E_f, \tilde{Y}_m)}{V(\tilde{Y}_m)} \\
&= \beta_m + \rho \frac{Cov(G_c, \tilde{Y}_m)}{V(\tilde{Y}_m)} + \underbrace{\beta_f \frac{Cov(Y_f, \tilde{Y}_m)}{V(\tilde{Y}_m)}}_{=0} + \omega_m \frac{Cov(E_m, \tilde{Y}_m)}{V(\tilde{Y}_m)} + \underbrace{\omega_f \frac{Cov(E_f, \tilde{Y}_m)}{V(\tilde{Y}_m)}}_{=0 \text{ (due to A2)}} \\
&= \beta_m + \rho \kappa_m \frac{Cov(G_m, \tilde{Y}_m)}{V(\tilde{Y}_m)} + \underbrace{\rho \kappa_f \frac{Cov(G_f, \tilde{Y}_m)}{V(\tilde{Y}_m)}}_{=0 \text{ (due to A2)}} + \omega_m \rho_{em} \frac{Cov(G_m, \tilde{Y}_m)}{V(\tilde{Y}_m)} + \omega_m \lambda_{em} \frac{Cov(C_m, \tilde{Y}_m)}{V(\tilde{Y}_m)} \\
&= \beta_m + \underbrace{\rho \kappa_m \frac{Cov(G_m, \tilde{Y}_m)}{V(\tilde{Y}_m)}}_{\text{Direct genetic transmission}} + \underbrace{\omega_m \rho_{em} \frac{Cov(G_m, \tilde{Y}_m)}{V(\tilde{Y}_m)}}_{\text{Genetic nurture}} + \underbrace{\omega_m \lambda_{em} \frac{Cov(C_m, \tilde{Y}_m)}{V(\tilde{Y}_m)}}_{\text{Environmental confounding}}
\end{aligned}
\tag{12}
$$

With $\tilde{Y}_m$ representing maternal education conditional on paternal education. The derivations are symmetrical for fathers.

A.1 Narrow genetic confounding

Narrow genetic confounding (NGC) measures the extent of the bias that is driven by direct genetic transmission. In our setting, is equal to:

$$
NGC = \rho \kappa_m \frac{Cov(G_m, \tilde{Y}_m)}{V(\tilde{Y}_m)} \tag{13}
$$

A.2 Broad genetic confounding

Broad genetic confounding is equal to the sum of direct genetic transmission and genetic nurture. However, since $\omega_m \lambda_{em}$ is unobserved, we calculate it as follows:

$$
\begin{aligned}
BGC &= \hat{\beta}_m - \hat{\beta}_m^{gc} \\
&= \beta_m + \rho\,\kappa_m \frac{Cov(G_m, \tilde{Y}_m)}{V(\tilde{Y}_m)} + \omega_m \rho_{em} \frac{Cov(G_m, \tilde{Y}_m)}{V(\tilde{Y}_m)} + \omega_m \lambda_{em} \frac{Cov(C_m, \tilde{Y}_m)}{V(\tilde{Y}_m)} \\
&\quad - \left(\beta_m + \underbrace{\rho\,\kappa_m \frac{Cov(G_m, \tilde{Y}_m^{gc})}{V(\tilde{Y}_m^{gc})}}_{=0} + \underbrace{\omega_m \rho_{em} \frac{Cov(G_m, \tilde{Y}_m^{gc})}{V(\tilde{Y}_m^{gc})}}_{=0} + \omega_m \lambda_{em} \frac{Cov(C_m, \tilde{Y}_m^{gc})}{V(\tilde{Y}_m^{gc})} \right) \\
&= \rho\,\kappa_m \frac{Cov(G_m, \tilde{Y}_m)}{V(\tilde{Y}_m)} + \omega_m \rho_{em} \frac{Cov(G_m, \tilde{Y}_m)}{V(\tilde{Y}_m)} + \omega_m \lambda_{em} \left(\frac{Cov(C_m, \tilde{Y}_m)}{V(\tilde{Y}_m)} - \frac{Cov(C_m, \tilde{Y}_m^{gc})}{V(\tilde{Y}_m^{gc})} \right) \\
&\approx \rho\,\kappa_m \frac{Cov(G_m, \tilde{Y}_m)}{V(\tilde{Y}_m)} + \omega_m \rho_{em} \frac{Cov(G_m, \tilde{Y}_m)}{V(\tilde{Y}_m)}
\end{aligned} \tag{14}
$$

With $\tilde{Y}_m^{gm}$ representing maternal education conditional on paternal education, father and mother genetic endowments. The derivations are symmetrical for fathers.

B Comparison to other methods

B.1 Controlling for the child's genetic endowments

One obvious alternative to our approach of controlling for the parental genetic endowments is to control for the child's genetic endowments. Figure 2 illustrates these relationships in a Directed Acyclical Graph (DAG).

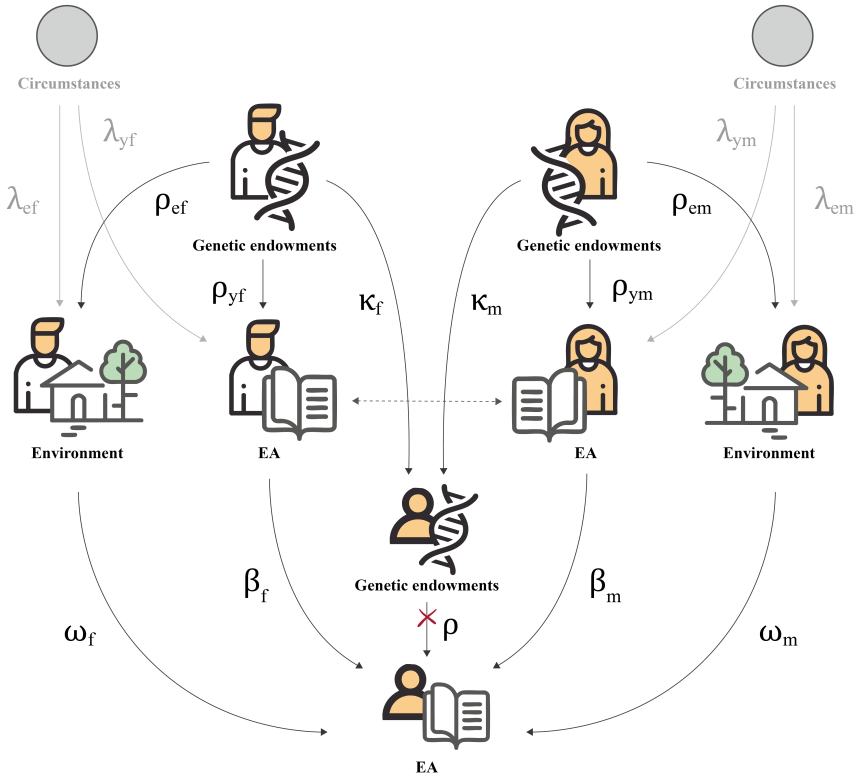

Figure 2 Directed Acyclic Graph (DAG) representing the causal paths between our interest variables.

The estimated model then takes the form

$$Y_c = \alpha + \rho G_c + \beta_m Y_m + \beta_f Y_f + \varepsilon_c \tag{15}$$

Under Assumption 2 (see Section 2), the estimated coefficient of equation (15) is given by

$$\begin{aligned}\hat{\beta}_m^{B.1} &= \frac{Cov(Y_c, \tilde{Y}_m^g)}{V(\tilde{Y}_m^g)} = \frac{Cov(\rho G_c + \beta_m Y_m + \beta_f Y_f + \omega_m E_m + \omega_f E_f, \tilde{Y}_m^g)}{V(\tilde{Y}_m^g)} \\ &= \beta_m + \underbrace{\rho \frac{Cov(G_c, \tilde{Y}_m^g)}{V(\tilde{Y}_m^g)}}_{=0} + \underbrace{\beta_f \frac{Cov(Y_f, \tilde{Y}_m^g)}{V(\tilde{Y}_m^g)}}_{=0} + \omega_m \frac{Cov(E_m, \tilde{Y}_m^g)}{V(\tilde{Y}_m^g)} + \underbrace{\omega_f \frac{Cov(E_f, \tilde{Y}_m^g)}{V(\tilde{Y}_m^g)}}_{=0 \text{ (due to A2)}} \\ &= \beta_m + \omega_m \rho_{em} \frac{Cov(G_m, \tilde{Y}_m^g)}{V(\tilde{Y}_m^g)} + \omega_m \lambda_{em} \frac{Cov(C_m, \tilde{Y}_m^g)}{V(\tilde{Y}_m^g)} \end{aligned} \tag{16}$$

With $\tilde{Y}_m^g$ representing maternal education conditional on paternal education and offspring's genetic endowments. It is important to note that controlling for the child's genetic endowments does not completely erase genetic confounding as in equation (8). Instead, the second term on the RHS shows that genetic nurture is not completely controlled for as the child's genetic endowments G_c exhibit a correlation of only 0.5 in expectation with the maternal genetic endowments G_m. As a result, taking differences with the coefficient of the simple association in equation (5) yields:

$$\begin{aligned}\hat{\beta}_m - \hat{\beta}_m^{B.1} &= \beta_m + \rho \kappa_m \frac{Cov(G_m, \tilde{Y}_m)}{V(\tilde{Y}_m)} + \omega_m \rho_{em} \frac{Cov(G_m, \tilde{Y}_m)}{V(\tilde{Y}_m)} + \omega_m \lambda_{em} \frac{Cov(C_m, \tilde{Y}_m)}{V(\tilde{Y}_m)} \\ &\quad - \left(\beta_m + \omega_m \rho_{em} \frac{Cov(G_m, \tilde{Y}_m^g)}{V(\tilde{Y}_m^g)} + \omega_m \lambda_{em} \frac{Cov(C_m, \tilde{Y}_m^g)}{V(Y_m | Y_f, G_c)} \right) \\ &= \underbrace{\rho \kappa_m \frac{Cov(G_m, \tilde{Y}_m)}{V(\tilde{Y}_m)}}_{\text{Direct genetic transmission}} + \underbrace{\omega_m \rho_{em} \left(\frac{Cov(G_m, \tilde{Y}_m)}{V(\tilde{Y}_m)} - \frac{Cov(G_m, \tilde{Y}_m^g)}{V(\tilde{Y}_m^g)} \right)}_{\text{Genetic nurture (fraction)}} \\ &\quad + \underbrace{\omega_m \lambda_{em} \left(\frac{Cov(C_m, \tilde{Y}_m)}{V(\tilde{Y}_m)} - \frac{Cov(C_m, \tilde{Y}_m^g)}{V(\tilde{Y}_m^g)} \right)}_{\text{Environmental confounding (residual)}} \end{aligned} \tag{17}$$

Controlling for the child's genetic endowments to account for direct genetic transmission or narrow genetic confounding captures two additional components: a residual of genetic nurture and a residual of environmental confounding. Whereas the latter residual is similar in nature to our broad genetic confounding estimate in equation (9), and plausibly small, the genetic nurture bias term is likely larger. Given that the correlation between G_m and Y_m to be 0.5, it is expected that the magnitude of the correlation halves once we residualize Y_m with respect to G_m. In contrast, the correlation between C_m and Y_m is environmental in nature such that we don't expect that residualizing with respect to G_m will significantly alter this correlation. The latter is verified empirically in Appendix F.3. As such, the genetic nurture term in equation (17) can be quantitatively meaningful and renders the estimated difference hard to interpret.

B.2 Adoption Studies

Adoption studies exploit the lack of genetic relationship between parents and their adoptive children. These studies rely on a variation of Assumption 1 (see Section 2), that adoptees are randomly assigned, such that there is no correlation between parental genetic endowments and the adopted child's genetic endowments ($Cov(G_p, G_c) = 0$). In addition, random assignment of adoptees also ensures that the environment, education, and genetic endowments of biological parents are uncorrelated with the educational attainment of adoptive parents. Specifically, this entails that $Cov(Y_p, Y_p^b) = 0$, $Cov(Y_p, G_p^b) = 0$, and $Cov(Y_p, E_p^b) = 0$, where b denotes the biological parent. Adoption studies also rely on Assumption 2, which assumes that assortative mating occurs only based on educational attainment.

Figure 3 illustrates these relationships in a Directed Acyclical Graph (DAG).

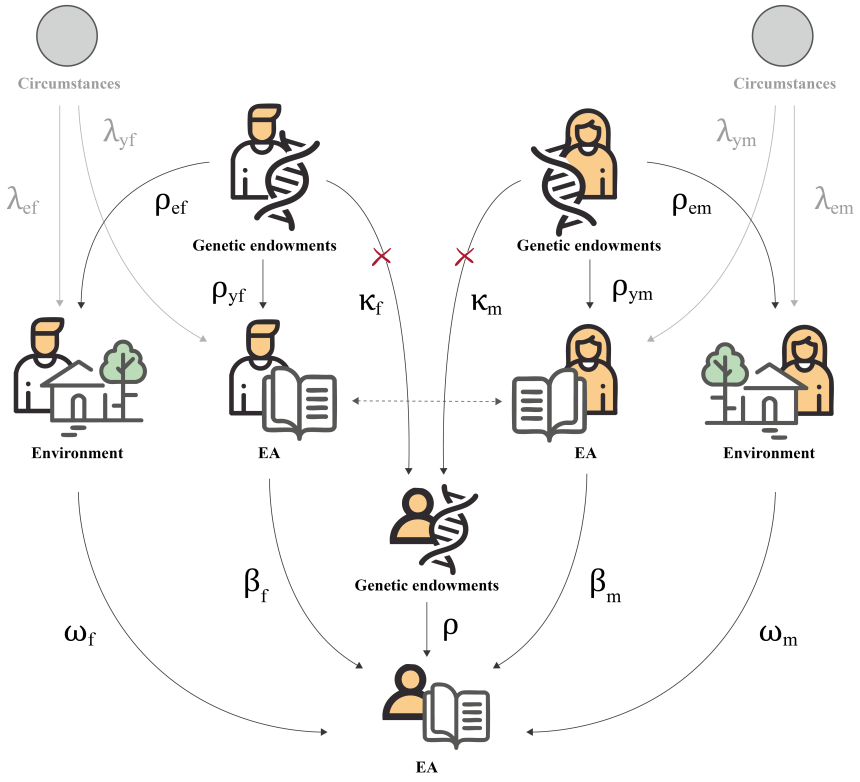

Figure 3 Directed Acyclic Graph (DAG) representing the causal paths between our interest variables.

In adoption studies, adopted children have two sets of parents: biological ones and adoptive ones. Educational attainment of the child is influenced by both:

$$Y_c = \alpha + \rho G_c + \beta_m Y_m + \beta_f Y_f + \beta_m^b Y_m^b + \beta_f^b Y_f^b + \omega_m E_m + \omega_f E_f + \omega_m^b E_m^b + \omega_f^b E_f^b + e_c, \tag{18}$$

where b denotes the biological parent. Assumption 1 ensures that biological parents' education and environment do not bias the β estimates. However it is important to notice that β and ω in this context only capture the impact of adoptive parents on the child's outcomes *after adoption*. Biological parents influence the in-utero environment, while foster care systems and/or biological parents may shape aspects of the early childhood environment. Consequently, β and ω are expected to differ between adopted and biological children.

$$\begin{aligned}
\hat{\beta}_m^{B.2} &= \frac{Cov(Y_c,\tilde{Y}_m)}{V(\tilde{Y}_m)} = \frac{Cov(\rho G_c+\beta_m Y_m+\beta_f Y_f+\beta_m^b Y_m^b+\beta_f^b Y_f^b+\omega_m E_m+\omega_f E_f+\omega_m^b E_m^b+\omega_f^b E_f^b,\tilde{Y}_m)}{V(\tilde{Y}_m)} \\
&= \beta_m + \underbrace{\rho\frac{Cov(G_c,\tilde{Y}_m)}{V(\tilde{Y}_m)}}_{\text{=0 (due to A1)}} + \underbrace{\beta_f\frac{Cov(Y_f,\tilde{Y}_m)}{V(\tilde{Y}_m)}}_{\text{=0}} + \underbrace{\beta_m^b\frac{Cov(Y_m^b,\tilde{Y}_m)}{V(\tilde{Y}_m)}+\beta_f^b\frac{Cov(Y_f^b,\tilde{Y}_m)}{V(\tilde{Y}_m)}}_{\text{=0 (due to A1)}} + \omega_m\frac{Cov(E_m,\tilde{Y}_m)}{V(\tilde{Y}_m)} + \underbrace{\omega_f\frac{Cov(E_f,\tilde{Y}_m)}{V(\tilde{Y}_m)}}_{\text{=0 (due to A2)}} \\
&= \beta_m + \omega_m\rho_{em}\frac{Cov(G_m,\tilde{Y}_m)}{V(\tilde{Y}_m)} + \omega_m\lambda_{em}\frac{Cov(C_m,\tilde{Y}_m)}{V(\tilde{Y}_m)} \qquad (19)
\end{aligned}$$

With $\tilde{Y}_m$ representing maternal education conditional on the father's education. Equation (19) illustrates that the association between an adopted child's outcomes and their adoptive parents' outcomes reflects three components: the causal effect of parental education on child outcomes, genetic nurture, and environmental confounding.

To estimate the effect of narrow genetic confounding, it is standard practice in adoption studies to subtract the β estimated in the adoptive children sample to the β^b estimated in the biological children sample. This comparison can be conducted within families (e.g. Sacerdote, 2007), or between different families (e.g., Plug, 2004). In both cases, differences between these estimates may arise due to the removal of direct genetic transmission, but also due differences in the causal effects of parental education, and environments on childhood educational attainment due to absence of parental in-utero and early childhood influence; $\beta^b \neq \beta$ and $\omega^b \neq \omega$:

$$\begin{aligned}
\hat{\beta}_m^b - \hat{\beta}_m^{B.2} &= \beta_m^b + \rho\kappa_m\frac{Cov(G_m,\tilde{Y}_m)}{V(\tilde{Y}_m)} + \omega_m^b\rho_{em}\frac{Cov(G_m,\tilde{Y}_m)}{V(\tilde{Y}_m)} + \omega_m^b\lambda_{em}\frac{Cov(C_m,\tilde{Y}_m)}{V(\tilde{Y}_m)} \\
&\quad - \left(\beta_m + \omega_m\rho_{em}\frac{Cov(G_m,\tilde{Y}_m)}{V(\tilde{Y}_m)} + \omega_m\lambda_{em}\frac{Cov(C_m,\tilde{Y}_m)}{V(\tilde{Y}_m)}\right) \\
&= \underbrace{\left(\beta_m^b-\beta_m\right)}_{\Delta\text{ causal effect}} + \underbrace{\rho\kappa_m\frac{Cov(G_m,\tilde{Y}_m)}{V(\tilde{Y}_m)}}_{\text{direct genetic transmission}} + \underbrace{\rho_{em}\frac{Cov(G_m,\tilde{Y}_m)}{V(\tilde{Y}_m)}(\omega_m^b-\omega_m)}_{\Delta\text{ genetic nurture}} + \underbrace{\lambda_{em}\frac{Cov(C_m,\tilde{Y}_m)}{V(\tilde{Y}_m)}(\omega_m^b-\omega_m)}_{\Delta\text{ environmental confounding}}
\end{aligned} \qquad (20)$$

Adoption studies aim to eliminate narrow genetic confounding from the parent-child education association. They depend on similar assumptions compared to other methods. However, by comparing adoptive and biological parents they are comparing parents whose influence on their children is inherently different due to the

lack of exposure of adopted children to the in-utero and early childhood environment of the adoptive parents.

B.3 Donor conceived children

A recent alternative to adoption studies has been to exploit donor conceived children (Rasmussen et al., 2024). In this case, a donor provides an egg or sperm to the parent such that the genetic transmission between at least one of the parents and child is broken. Compared with regular adoption designs, the issue of the biological parent influencing the child's prenatal and early childhood environment is solved, which is a major advantage. However, since this donor will only replace one parent in genetic transmission, the educational attainment of one parent will still correlate with the genetic endowments of their partner. This means that controlling for the partner's educational attainment and assuming Assumption 2 (see Section 2) is necessary. This design also requires a variation of Assumption 1, that parents do not select the donor based on relevant characteristics, ensuring no correlation between the genetic endowments of the non-biological parent (here, the mother) and the child, $Cov(G_m, G_c) = 0$. Figure 4 shows how the link between one parent and offspring genetic endowments is broken.

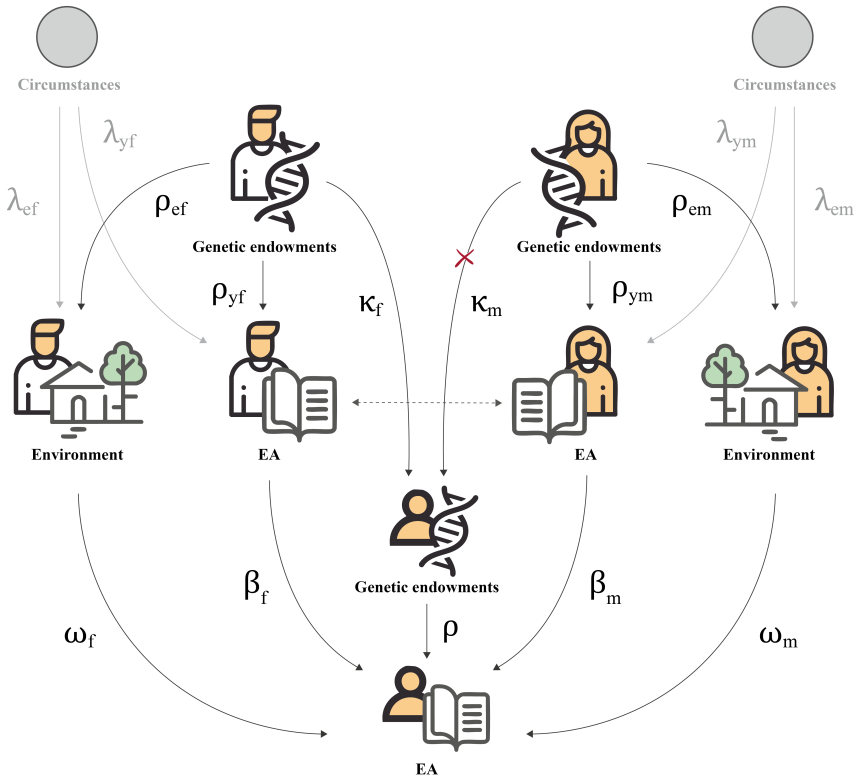

Figure 4 Directed Acyclic Graph (DAG) representing the causal paths between our interest variables, assuming an egg donor.

The derivations are similar to the ones from adoption studies B.2. Under a variation of Assumption 1 and Assumption 2, direct genetic transmission is effectively controlled for. However, obtaining an estimate of narrow genetic confounding requires comparing $\beta's$ between biological and donor-conceived children. In this case, donors are not expected to influence in-utero or early childhood environment. Nevertheless, it is possible that parents who resort to IVF might be different than those who have biological children in ways we cannot measure and hence, it is possible that the coefficients from the two samples are not comparable in expectation and do not cancel out (see equation 20).

B.4 Children of Twins

The children of twins design exploits the fact that identical (homozygotic) twins share the same genetic material. By comparing the outcomes of children of identical twins, the genetic endowments from one parent is controlled for. Consequently, this approach accounts for the three mechanisms stemming from parental genetic endowments: genetic transmission, parental educational attainment, and environmental factors. This design relies on Assumption 1 and 2 (see Section 2), which state that maternal genetic endowments do not correlate with maternal circumstances, and that assortative mating is only at the educational attainment level (provided partner educational attainment is controlled for). Figure 5 illustrates this relationship, depicting the case where the mother belongs to an identical twin pair.

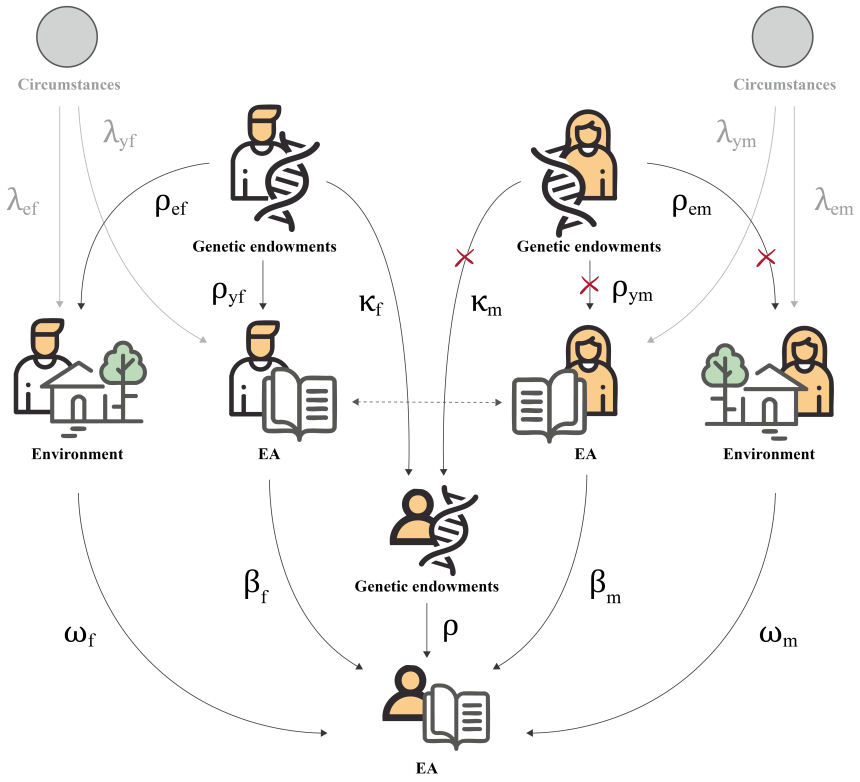

Figure 5 Directed Acyclic Graph (DAG) representing the causal paths between our interest variables.

In the children of twins design, the educational attainment of the child Y_c is replaced by $Y_c^1 - Y_c^2$, which represents the difference in outcomes between the children of the twin mothers, $1,2$. Consequently, the differences in maternal education and environment reflect the impact of distinct circumstances alone: $\Delta Y_m = \Delta\alpha_{ym} + \lambda_{ym}\Delta C_m + \Delta e_m$ and $\Delta E_m = \Delta\alpha_{em} + \lambda_{em}\Delta C_m + \Delta\eta_m$ as $\Delta G_m = \varepsilon_m$, where ε_m is equal to *de novo* mutations.

In terms of the estimator,

$$
\begin{aligned}
\hat{\beta}_m^{B.4} &= \frac{Cov(\Delta Y_c, \Delta\tilde{Y}_m)}{V(\Delta\tilde{Y}_m)} = \frac{Cov(\rho\Delta G_c + \beta_m\Delta Y_m + \beta_f\Delta Y_f + \omega_m\Delta E_m + \omega_f\Delta E_f, \Delta\tilde{Y}_m)}{V(\Delta\tilde{Y}_m)} \\
&= \beta_m + \rho\frac{Cov(\Delta G_c, \Delta\tilde{Y}_m)}{V(\Delta\tilde{Y}_m)} + \omega_m\frac{Cov(\Delta E_m, \Delta\tilde{Y}_m)}{V(\Delta\tilde{Y}_m)} \\
&= \beta_m + \underbrace{\rho\lambda_{ym}\frac{Cov(\Delta G_c, (\Delta C_m|\Delta Y_f))}{V(\Delta\tilde{Y}_m)}}_{\text{=0 (due to A1)}} + \omega_m\lambda_{em}\frac{Cov(\Delta C_m, \Delta\tilde{Y}_m)}{V(\Delta\tilde{Y}_m)} \\
&= \beta_m + \omega_m\lambda_{em}\frac{Cov(\Delta C_m, \Delta\tilde{Y}_m)}{V(\Delta\tilde{Y}_m)}
\end{aligned}
\tag{21}
$$

The children of twins design subsequently compares the OLS estimate to the differences estimator, within the same sample of twins:

$$\begin{aligned}\hat{\beta}_m - \hat{\beta}_m^{B.4} &= \beta_m + \rho\,\kappa_m \frac{Cov(G_m, \tilde{Y}_m)}{V(\tilde{Y}_m)} + \omega_m \rho_{em} \frac{Cov(G_m, \tilde{Y}_m)}{V(\tilde{Y}_m)} + \omega_m \lambda_{em} \frac{Cov(C_m, \tilde{Y}_m)}{V(\tilde{Y}_m)} \\ &\quad - \left(\beta_m + \omega_m \lambda_{em} \frac{Cov(\Delta C_m, \Delta \tilde{Y}_m)}{V(\Delta \tilde{Y}_m)} \right) \\ &= \underbrace{\rho\,\kappa_m \frac{Cov(G_m, \tilde{Y}_m)}{V(\tilde{Y}_m)}}_{\text{direct genetic transmission}} + \underbrace{\omega_m \rho_{em} \frac{Cov(G_m, \tilde{Y}_m)}{V(\tilde{Y}_m)}}_{\text{genetic nurture}} + \underbrace{\omega_m \lambda_{em} \left(\frac{Cov(C_m, \tilde{Y}_m)}{V(\tilde{Y}_m)} - \frac{Cov(\Delta C_m, \Delta \tilde{Y}_m)}{V(\Delta \tilde{Y}_m)} \right)}_{\text{environmental confounding (fraction)}} \end{aligned} \tag{22}$$

This design estimates broad genetic confounding by capturing both direct genetic transmission and genetic nurture effects. It also accounts for a portion of environmental confounding. The portion of environmental confounding is captured since the difference between the between and within family association is not necessarily zero. The between-family term $\frac{Cov(C_m, \tilde{Y}_m)}{V(\tilde{Y}_m)}$ captures the association between all circumstances and education. In contrast, the within-family association $\frac{Cov(\Delta C_m, \Delta \tilde{Y}_m)}{V(\Delta \tilde{Y}_m)}$ isolates the individual-specific circumstances effect, removing shared family circumstances. For instance, if two twins receive slightly different amounts of tutoring, but their overall educational attainment is mostly shaped by shared family resources and background, this could lead to a difference between these two measures. The reason is that tutoring correlates with family resources and background, and hence the first term would capture a large correlation – stemming from the association between tutoring and family resources – whereas the second term would capture a small correlation – due to the small causal effect of tutoring.

C A primer on genetics

In 2003, a complete sequencing of the human was achieved for the first time. Coupled with an exponential decrease of the cost of genotyping individuals, this led to the creation of several data sets that contained individual genetic information. The availability of data allowed researchers to identify linkages between a person's genetic variants and important life outcomes such as health, personality, and education (Benjamin et al., 2012; Visscher et al., 2017). Virtually all human traits are highly "polygenic" (Visscher et al., 2008; Chabris et al., 2015). That is, there is no 'gene for' these outcomes; instead, the vast majority of human outcomes are affected by many alleles, each with very small effect sizes. Recent advances in the genetics field such as the completion of the Human Genome Project in the early 2000s and the development of inexpensive genotyping chips have made it possible to identify links between a person's genome and socially relevant outcomes such as health and education (Beauchamp et al., 2011; Visscher et al., 2017).

The human genome consists of more than 3.2 billion nucleotides located on 23 pairs of chromosomes (Lehrer and Ding, 2017). These nucleotides come in four varieties: adenine (A), guanine (G), cytosine (C) and thymine (T). Approximately 99.6 percent of the nucleotides are identical between two randomly selected individuals (Kidd et al., 2008). There are certain positions or loci where individuals have different nucleotides. The most common type of such genetic variation is called a single nucleotide polymorphism (SNP). SNPs constitute the main source of genetic differences between individuals. At each SNP location there can be two different nucleotides. It is common to measure SNPs by counting the number of minor alleles (the nucleotide that occurs least frequently in the population) that an individual carries. Hence, a SNP can take the values 0, 1 or 2. It is common to sum up the number of reference alleles because virtually all genetic variants contribute additively to genetically influenced traits (Pazokitoroudi et al., 2021). In some rare cases, a difference at a specific position on a chromosome can single-handedly lead to a disease: Huntington's disease is an example. However, the vast majority of human (behavioral) traits are polygenic, meaning they are influenced by multiple genetic polymorphisms, each contributing with a tiny effect (Chabris et al., 2015). A polygenic score is constructed by adding up the individual SNPs, where each SNP is weighted by the strength of the association between the SNP and the outcome variables as estimated in a genome-wide association study (GWAS) (Dudbridge, 2013). The underlying rationale is that based on GWAS results, you can assign weights of relative importance to each SNP. Then, with a polygenic score, one can exploit the joint predictive power of multiple SNPs for a particular outcome.

C.1 Heritability and predictive power of PGIs

Before genotyping of humans was a possibility, research relied on twin studies to quantify the heritability of educational attainment. Heritability is defined as the fraction of trait variation in a population due to genetic

inheritance. Twin studies estimate heritability comparing monozygotic to dizygotic twins. Since monozygotic twins are genetically identical, whereas non-identical twins share on average half of their genetic material, greater similarity of identical over non-identical twins is evidence for a contribution of genetic variation to trait variation. However, the twin design makes several assumptions, most importantly that there is no difference in shared environment between identical and non-identical twins. Since this assumption has been called into question (Joseph, 2002; Beckwith and Morris, 2008; Dalmaijer, 2020) it remains controversial whether twin studies have overestimated heritability for human traits (Felson, 2014).

When genotyping individuals became possible, researchers started to question whether estimating heritability from directly measuring genetic variants was possible. This type of estimate is referred to as SNP-based heritability (Yang et al., 2010). SNP-based heritability is defined as the fraction of phenotypic variance that can be attributed to variation in the additive effects of common genetic variants. In 2010, the Genomic Relatedness Restricted Maximum Likelihood (GREML) (Yang et al., 2011) methodology was developed. GREML estimates the variance explained by the SNPs by accessessing how similar are the genomes of individuals with similar traits, without the need of estimating an individual weight for each SNP. GREML restricts the analysis to distantly related individuals to avoid bias due to to environmental effects shared between close relatives and genetic interactions (Yang et al., 2010, 2017). Further, at the time, the GREML methodology could only include genetic variants that are common in the population (Yang et al., 2017; Wainschtein et al., 2022). At that point SNP-based heritability was much lower than twin-based heritability estimates, which was referred to as the missing heritability problem (Young, 2019).

Many different explanations for the 'missing heritability' have been proposed (Eichler et al., 2010). One of them is that twin studies have overestimated heritability or that they lack external validity. A second type of explanations claims that SNP-based heritability only captures additive effects and common variants whereas complex traits are likely to be affects by many rare variants. To respond to the last proposed explanation, the GREML methodology was extended to include rarer genetic variations inferred by imputation (Yang et al., 2015) and subsequently it was further extended to include high quality whole genome sequence (WGS) data (Wainschtein et al., 2022). This significantly increased the SNP-based heritability estimates of height from 0.49 to 0.56 and 0.70, well in line with twin estimates of 0.7-0.8 (Young, 2019). It also increased the heritability estimates of BMI from 0.21 to 0.29, still below the twin heritability estimates of 0.4-0.6. The GREML methodology with high quality whole genome data has not been extended to educational attainment yet.

To sum up, the true size of heritability of traits including of educational attainment is an ongoing debate. On the one hand, twin-based heritability might be overestimated and might not be externally valid. On the other, SNP-based heritability only captures additive effects, and imputed wide genome data. The SNP-based heritability for educational attainment is estimated to be between 22 and 28% (Rietveld et al., 2013; Davies et al., 2016; Tropf et al., 2017). However, the most recent EA PGI predicts up to 16% of the variation in

educational attainment (Okbay et al., 2022). This implies that PGI-based heritability is currently lower than SNP-based heritability. van Kippersluis et al. (2023) suggest that measurement error in the PGIs is a key explanation for this difference and propose using two independent PGIs as instrument for each other, which provides a considerable increase in PGI-based heritability estimates. We will make use of this method to reduce the measurement error of our PGI.

It is clear from recent studies that the SNP-based heritability and PGI-based heritability do not necessarily stem from direct (or 'causal') genetic effects only. Remember that the PGI is constructed based on a GWAS, which is a univariate regression of the outcome on one SNP at a time. Since this SNP is inherited from parents, any influence of parental (or other family members) SNPs on the offspring outcome will bias the direct genetic effect of that SNP. These are called indirect genetic effects. Tackling the issue of indirect genetic effects for traits like educational attainment requires large samples of families coupled with whole genome high quality data (Young, 2019). Sib-Regression (Wray et al., 2019) or relatedness disequilibrium regression (RDR) (Young et al., 2018) are SNP-based heritability methods that take advantage of the random variation in relatedness between siblings in a family to estimate heritability with little bias from population stratification and environment. Compared to twin heritability estimates of 65% (Pedersen et al., 2002) and GREML heritability of 22-28% (Rietveld et al., 2013; Davies et al., 2016; Tropf et al., 2017), RDR estimates an heritability for educational attainment of 17% (Young et al., 2018), widening the gap between the twin- and SNP-based heritability estimates again. Some authors interpret this difference as evidence that twin based heritabilities are likely overestimated (Young, 2019).

D Details on PGI construction

D.1 GWAS

The UKBiobank (UKB) EA PGI was constructed using the GWAS summary statistics of Muslimova et al. (2025). The GWAS was performed using the fastGWA tool for Genome-wide Complex Trait Analysis (GCTA) developed by Jiang et al. (2019) in the UK Biobank data set. The full UKB GWAS discovery sample includes 392,771 individuals. The UKB discovery sample 1 and 2 were obtained by randomly splitting the full discovery sample into two samples of approximately 196,380 individuals. The full GWAS details are described in Muslimova et al. (2025).

The 23andMe EA PGI was constructed using the GWAS summary statistics made by 23andMe. The 23andMe sample includes 365,536 individuals. More details GWAS details are described in Lee et al. (2018).

D.2 LDpred weights

The EA PGI was constructed using the sum of the number of reference allele for each SNP multiplied by the effect size that was corrected for linkage disequilibrium using the software package LDpred (Vilhjálmsson et al., 2015). In LDpred, a random 30k subsample of the UKB was used as the coordination data set. The target data set consists of 19,999 individuals; 9,351 mothers, 8,927 children, and 1,721 fathers. A prior of 1 was set. For the LDpred weights constructed using the full UKB summary statistics, a 355-kb window and 1,065,139 HapMap 3 SNPs were used. For the weights constructed using the 23andMe summary statistics a 352-kb window and 1,057,143 HapMap 3 SNPs were used. For the UKB sample 1 and sample 2 a 355-kb window and 1,065,139 Hapmap 3 SNPs were used.

D.3 Imputation and PGI constructing

In order to expand our trio data set, we used SNIPAR, a software tool developed by Young et al. (2022) that exploits pedigree information to impute parental or sibling genotypes. We exclude variants with Hardy-Weinberg equilibrium exact test p-value below 1×10^{-6}, with missing genotyping rate larger than 5%, or with minor allele frequency of less than 5%. We also excluded 1 twin from each twin duo, and individuals with more than 5% missing genotype. This resulted in a data set with 16,276 individuals and 1,283 mother-father-child trios. The 5,513 mother-child pairs were used to impute the genome of 4,230 fathers, resulting in a genotyped sample of 5,513 trios (or 16,539 individuals). Finally, The LDpred weights described above were used to construct the PGIs in this expanded trios data set.

E Estimating narrow genetic confounding

To estimate narrow genetic confounding (see Section 2), we have to compute $\rho\kappa_m \frac{Cov(G_m,\tilde{Y}_m)}{V(\tilde{Y}_m)}$ for the mother, and $\rho\kappa_f \frac{Cov(G_f,\tilde{Y}_f)}{V(\tilde{Y}_f)}$ for the father, with $\tilde{Y}_m$ being maternal education conditional on the father's and vice versa for $\tilde{Y}_f$.

We estimate $\hat{\rho}$ as the coefficient of the child's EA PGI in a regression with the child's KS4 score as dependent variable and the parental EA PGIs as controls:

$$KS4 = \alpha + \rho PGI_c + \delta_m PGI_m + \delta_f PGI_f + \varepsilon \tag{23}$$

In this regression, we account for measurement error using ORIV with two independent PGIs for each of the three PGIs of children, mothers and fathers. Table 2 shows that ρ is estimated as 21.3 (OLS, column 4) and 39.8 (ORIV, column 7).

We know that κ_m and κ_f are 0.5 as both fathers and mothers transmit a half of their genetics to their offspring.

Finally, we estimate $\frac{Cov(G_m,\tilde{Y}_m)}{V(\tilde{Y}_m)}$ as the regression coefficient of EA_m in the regression of PGI_m on EA_m and EA_f, and $\frac{Cov(G_f,\tilde{Y}_f)}{V(\tilde{Y}_f)}$ as the regression coefficient of EA_f in the regression of PGI_f on EA_f and EA_m:

$$PGI_m = \mu_{0,m} + \mu_{m,m} EA_m + \mu_{f,m} EA_f + \psi_m \tag{24}$$

$$PGI_f = \mu_{0,f} + \mu_{m,f} EA_m + \mu_{f,f} EA_f + \psi_f \tag{25}$$

Equations 24 and 25 depict a simple OLS estimate, without any measurement error correction. Under this specification the estimates are given as $\hat{\mu}_{m,m} = 0.178$ and $\hat{\mu}_{f,f} = 0.104$.

In contrast to equation (23), the PGIs are now on the left-hand-side (24) and (25). Without standardization, measurement error on the left-hand-side does not cause a bias in the coefficients. However, when the dependent variables is measured with error *and* standardized, the resulting coefficients will be biased.

To see this, consider the case where we don't observe the true latent polygenic index PGI^*, but an estimated PGI that is measured with error:

$$PGI = PGI^* + \nu, \quad \nu \sim N\left(0, \sigma_\nu^2\right)$$

where we assume that the measurement error ν is classical. If – as is common in the literature – the observed

PGI is standardized to obtain PGI_{st}, it follows that:

$$\begin{aligned} PGI_{st} &= \frac{PGI^* + \nu - \mu_{PGI}}{\sqrt{\sigma^2_{PGI^*} + \sigma^2_\nu}} \\ &= \frac{PGI^* + \nu}{\sqrt{\sigma^2_{PGI^*} + \sigma^2_\nu}} \end{aligned} \tag{26}$$

since $\mu_{PGI} = 0$ in expectation.

If we now run the regression $PGI_{st} = \mu + \beta_{st} Y + \eta$, we get

$$\begin{aligned} \hat{\beta}_{st} &= \frac{Cov(PGI_{st}, Y)}{V(Y)} \\ &= \frac{Cov(\frac{PGI^* + \nu}{\sqrt{\sigma^2_{PGI^*} + \sigma^2_\nu}}, Y)}{\sigma^2_Y} \\ &= \frac{\beta \sigma^2_{PGI^*}}{\sqrt{\sigma^2_{PGI^*} + \sigma^2_\nu}} \Big/ \sigma^2_Y \\ &= \beta_{st} \frac{\sigma_{PGI^*}}{\sqrt{\sigma^2_{PGI^*} + \sigma^2_\nu}} \Big/ \sigma^2_Y \end{aligned} \tag{27}$$

If we have two independent standardized measures of the true PGI^*, each measured with independent errors that have the same variance

$$\begin{aligned} PGI_{1,st} &= \frac{PGI^* + \nu_1}{\sqrt{\sigma^2_{PGI^*} + \sigma^2_\nu}} \\ PGI_{2,st} &= \frac{PGI^* + \nu_2}{\sqrt{\sigma^2_{PGI^*} + \sigma^2_\nu}} \end{aligned} \tag{28}$$

Then

$$Cov(PGI_{1,st}, PGI_{2,st}) = Cov\Big(\frac{PGI^* + \nu_1}{\sqrt{\sigma^2_{PGI^*} + \sigma^2_\nu}}, \frac{PGI^* + \nu_2}{\sqrt{\sigma^2_{PGI^*} + \sigma^2_\nu}}\Big) = \frac{\sigma^2_{PGI^*}}{\sigma^2_{PGI^*} + \sigma^2_\nu} \tag{29}$$

And in turn,

$$\sqrt{Cov(PGI_{1,st}, PGI_{2,st})} = \frac{\sigma_{PGI^*}}{\sqrt{\sigma^2_{PGI^*} + \sigma^2_\nu}} \tag{30}$$

Hence, in practice we scale the standardized noisy PGI ex-ante by (30), such that

$$PGI^+ = \frac{PGI_{st}}{\sqrt{Cov(PGI_{1,st}, PGI_{2,st})}} = \frac{PGI^* + \nu}{\sigma_{PGI^*}} \tag{31}$$

In turn, instead of equations (24) and (25), we run the regressions

$$PGI_m^+ = \mu_{0,m} + \mu_{m,m} EA_m + \mu_{f,m} EA_f + \psi_m \tag{32}$$

$$PGI_f^+ = \mu_{0,f} + \mu_{m,f} EA_m + \mu_{f,f} EA_f + \psi_f \tag{33}$$

which gives the correct standardized estimate $\hat{\mu}_{m,m}$ for $\frac{Cov(G_m,\tilde{Y}_m)}{V(\tilde{Y}_m)}$ and $\hat{\mu}_{f,f}$ for $\frac{Cov(G_f,\tilde{Y}_f)}{V(\tilde{Y}_f)}$. The resulting estimates are given as $\hat{\mu}_{m,m} = 0.245$ and $\hat{\mu}_{f,f} = 0.147$.

F Relaxing assumptions in the baseline model

In this section, we relax the two assumptions in our baseline model and investigate whether their removal affects our results.

F.1 The model

In our baseline model, we adopt a two-generation model, assuming that maternal and paternal circumstances are uncorrelated with their genes. However, parental circumstances are likely influenced by grandparental or other relatives' genetic endowments through genetic nurture (e.g., Kong et al., 2018). Additionally, parental genes may correlate with their circumstances due to population stratification (e.g. Young et al., 2019). The idea of population stratification is that, over generations, genetic variation (allele frequencies) starts to differ across population subgroups. This happens when mating patterns are shaped by factors such as geographic proximity or socioeconomic background, rather than occurring randomly. If these same population subgroups in turn have different cultural habits, this could lead to an association between genes and environmental circumstances. To model this, we decompose parental circumstances into two components: environmental circumstances, EC, and genetic driven circumstances, GC. Parental genetic endowments G_p are associated with GC_p through genetic nurture, and with EC_p through population stratification.

Our baseline specification also assumes that parents assort based solely on educational attainment. In reality, it is likely that parents sort on other characteristics. The subsequent subsections relax Assumptions 1 and 2. First, we explore their implications for the narrow genetic confounding estimate, followed by a similar analysis for the broad genetic confounding estimate. In this section, we adopt a different notation for ease of derivation: $A|B$ instead of $\tilde{A}$ to make it clearer what is being conditioned upon.

$$Y_c = \alpha + \rho G_c + \beta_m Y_m + \beta_f Y_f + \omega_m E_m + \omega_f E_f + e_c \tag{34}$$

$$G_c = \alpha_G + \kappa_m G_m + \kappa_f G_f + \varepsilon_G \tag{35}$$

$$Y_p = \alpha_{yp} + \rho_{yp} G_p + \lambda_{yp} EC_p + \zeta_{yp} GC_p + e_p \quad \text{with } p = m, f \tag{36}$$

$$E_p = \alpha_{ep} + \rho_{ep} G_p + \lambda_{ep} EC_p + \zeta_{ep} GC_p + \eta_p \quad \text{with } p = m, f \tag{37}$$

The OLS estimator of equation (5) when the true data generating process is given by equations (34) to (37)

is

$$
\begin{aligned}
\hat{\beta}_m &= \frac{Cov(Y_c,(Y_m|Y_f))}{V(Y_m|Y_f)} \\
&= \frac{Cov(\rho G_c+\beta_m Y_m+\beta_f Y_f+\omega_m E_m+\omega_f E_f,(Y_m|Y_f))}{V(Y_m|Y_f)} \\
&= \beta_m+\rho\frac{Cov(G_c,(Y_m|Y_f))}{V(Y_m|Y_f)}+\omega_m\frac{Cov(E_m,(Y_m|Y_f))}{V(Y_m|Y_f)}+\omega_f\frac{Cov(E_f,(Y_m|Y_f))}{V(Y_m|Y_f)} \\
&= \beta_m+\underbrace{\rho\kappa_m\frac{Cov(G_m,(Y_m|Y_f))}{V(Y_m|Y_f)}}_{\text{Estimated NGC}}+\rho\kappa_f\frac{Cov(G_f,(Y_m|Y_f))}{V(Y_m|Y_f)} \\
&\quad+\omega_m\rho_{em}\frac{Cov(G_m,(Y_m|Y_f))}{V(Y_m|Y_f)}+\omega_m\lambda_{em}\frac{Cov(EC_m,(Y_m|Y_f))}{V(Y_m|Y_f)}+\omega_m\zeta_{em}\frac{Cov(GC_m,(Y_m|Y_f))}{V(Y_m|Y_f)} \\
&\quad+\omega_f\rho_{ef}\frac{Cov(G_f,(Y_m|Y_f))}{V(Y_m|Y_f)}+\omega_f\lambda_{ef}\frac{Cov(EC_f,(Y_m|Y_f))}{V(Y_m|Y_f)}+\omega_f\zeta_{ef}\frac{Cov(GC_f,(Y_m|Y_f))}{V(Y_m|Y_f)}
\end{aligned}
\tag{38}
$$

F.2 Narrow genetic confounding

The second term on the right-hand side ("Estimated NGC") is how we calculate narrow genetic confounding (NGC) in our baseline model; the amount of genetic confounding attributable to direct genetic transmission. In our baseline model, we assume that parental genes are independent of parental circumstances. Under this assumption, the term $\frac{Cov(G_m,(Y_m|Y_f))}{V(Y_m|Y_f)}$ can be estimated as the regression coefficient of Y_m on G_m in a regression of G_m on Y_m and Y_f. If we relax this assumption (i.e., when parental genes are correlated to parental circumstances), the estimator no longer captures narrow genetic confounding only. G_m is now correlated to the environmental circumstances EC_m through population stratification, and to genetic circumstances GC_m due to genetic nurture of other relatives. In this setting, population stratification merely associates with maternal education – mating patterns generate a spurious association between genes and a trait – whereas genetic nurture actually influences maternal education Y_m (see equation 36) - relatives' genes causally modify one's educational outcome.

To see the logic, consider the narrow genetic confounding term, where for the sake of simplicity, we assume that Y_m is independent of Y_f:

$$
\begin{aligned}
\text{Genetic transmission path} &= \rho\kappa_m\frac{Cov(G_m,Y_m)}{V(Y_m)} \\
&= \rho\kappa_m\frac{Cov(G_m,\rho_{ym}G_m+\lambda_{ym}EC_m+\zeta_{ym}GC_m)}{V(Y_m)} \\
&= \underbrace{\rho\kappa_m\frac{\rho_{ym}}{V(Y_m)}}_{\text{NGC}}+\underbrace{\rho\kappa_m\lambda_{ym}\frac{Cov(G_m,EC_m)}{V(Y_m)}}_{\text{Population stratification}}+\underbrace{\rho\kappa_m\zeta_{ym}\frac{Cov(G_m,GC_m)}{V(Y_m)}}_{\text{Genetic nurture}}
\end{aligned}
\tag{39}
$$

Hence, our narrow genetic confounding estimator is biased by population structure and genetic nurture. Whereas bias stemming from population structure is likely small in ALSPAC (see F.3), we cannot rule out a moderate bias in our estimates from genetic nurture. To be able to calculate narrow genetic confounding we would have to calculate ρ_{ym}, this is, the causal impact of maternal (paternal) genes on their educational attainment. For this purpose, we would require a three generation data set or a sibling sample. Whereas our data set does not have three generations, it does contain a small subsection of mothers that happen to be sisters ($N = 195$).

Table F2 shows the results of an OLS regression explaining maternal educational attainment in ALSPAC, with and without sibling fixed-effects, and correcting for measurement error using IV. We obtain a ρ_{ym} estimate of 0.39.[20]

	OLS				IV			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
PGI mother (UKB)	0.311***		0.344*		0.362**		0.299	
	(0.086)		(0.146)		(0.127)		(0.198)	
PGI mother (23me)		0.254**		0.227		0.466***		0.479*
		(0.090)		(0.153)		(0.137)		(0.216)
Sibling fixed effects	No	No	Yes	Yes	No	No	Yes	Yes
R-squared	0.063	0.040	0.656	0.644	-	-	-	-
N	195	195	195	195	195	195	195	195
First stage F-stat -	-	-	-	-	72.901	72.901	31.716	31.716

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Table F2 Results of the OLS and IV regressions explaining the maternal years of education. Standard errors in parenthesis.

Using the variance of maternal years of education, which is equal to 3.063 (see Table 1), this gives NGC= 39.77*0.5*(0.389/3.063)=2.525 which corresponds to 18% of the mother–child association. This calculation suggests that parental genetic nurture introduces an upward bias in our baseline NGC estimates. The advantage of this approach is that it provides a flexible way to approximate the magnitude of this bias while relaxing some of our baseline assumptions. There are nevertheless two main limitations; first, we estimate ρ_{ym} in a small subsample of our data, which limits its external validity, and second, we are not able to do a similar exercise for fathers as there are no fathers who are brothers in ALSPAC.

[20] We weren't able to implement ORIV with sibling fixed effects, as demeaning at both the family level (to remove shared environmental factors) and at the polygenic index (PGI) level led to an induced negative correlation between the instruments. As such, both first and second stage coefficients were negative. In the raw data, PGIs are positively correlated across siblings, and standard within-family OLS yields a positive coefficient. This indicates that the negative first stage is a statistical artifact of combining ORIV with sibling fixed-effects, rather than a genuine feature of the data. As such, for the purpose of this exercise, we use the average of the two IV estimators 0.30 and 0.48 - Column 7 and 8 of Table F2.

F.3 Broad genetic confounding

In our baseline specification, we define broad genetic confounding as the sum of narrow genetic confounding and genetic nurture. Genetic nurture was defined as the influence of parental genes on the rearing environment of their offspring. Here, we expand the framework to include a third generation, the grandparents. In this expanded setting, genetic nurture also includes the influence of grandparental genes. Grandparental genetic nurture may influence grandchild's educational attainment either directly, by shaping their rearing environment, or indirectly, through the impact on their parents.[21] Direct genetic nurture from grandparents may occur, for example, when grandparents play an active role in raising their grandchildren, sharing caregiving responsibilities with the parents, such that their genes directly influence the rearing environment. Indirect genetic nurture arises when grandparental genes affect the genes and behaviors of the parents, who in turn create a different rearing environment for the child.

To understand the consequences for broad genetic confounding, rearrange equation (38):

$$
\begin{aligned}
\hat{\beta}_m &= \frac{Cov(Y_c,(Y_m|Y_f))}{V(Y_m|Y_f)} \\
&= \beta_m + \rho\frac{Cov(G_c,(Y_m|Y_f))}{V(Y_m|Y_f)} + \omega_m\frac{Cov(E_m,(Y_m|Y_f))}{V(Y_m|Y_f)} + \omega_f\frac{Cov(E_f,(Y_m|Y_f))}{V(Y_m|Y_f)} \\
&= \beta_m + \underbrace{\rho\,\kappa_m\frac{Cov(G_m,(Y_m|Y_f))}{V(Y_m|Y_f)}}_{\text{Estimated NGC}} + \underbrace{\omega_m\rho_{em}\frac{Cov(G_m,(Y_m|Y_f))}{V(Y_m|Y_f)}}_{\text{Parental Genetic Nurture}} + \underbrace{\omega_m\zeta_{em}\frac{Cov(GC_m,(Y_m|Y_f))}{V(Y_m|Y_f)}}_{\text{Grandparental Genetic Nurture}} \\
&\quad + \underbrace{\omega_m\lambda_{em}\frac{Cov(EC_m,(Y_m|Y_f))}{V(Y_m|Y_f)}}_{\text{Environmental confounding + population stratification}} \\
&\quad + \underbrace{\left(\omega_f\rho_{ef}+\rho\,\kappa_f\right)\frac{Cov(G_f,(Y_m|Y_f))}{V(Y_m|Y_f)} + \omega_f\lambda_{ef}\frac{Cov(EC_f,(Y_m|Y_f))}{V(Y_m|Y_f)} + \omega_f\zeta_{ef}\frac{Cov(GC_f,(Y_m|Y_f))}{V(Y_m|Y_f)}}_{\text{Conditional Assortative Mating}}
\end{aligned}
\tag{40}
$$

In a three-generation model, broad genetic confounding is still the sum of direct genetic transmission and genetic nurture, except that in a three generation model, genetic nurture can be at the parent level, or at the grandparental level, which can run directly or indirectly. The next step is to derive the estimator of β_m when G_m and G_f are added as controls.

[21] In practice, broad genetic confounding encompasses all confounding attributable to the causal effects of genes of relatives on the child's educational attainment, irrespective of the family member involved or the pathway through which these effects operate. For this analysis, however, we interpret it as the influence of grandparents.

$$
\begin{aligned}
\hat{\beta}_m^2 &= \frac{Cov(Y_c,(Y_m|Y_f,G_m,G_f))}{V(Y_m|Y_f,G_m,G_f)} \\
&= \beta_m + \underbrace{\rho\,\kappa_m \frac{Cov(G_m,(Y_m|Y_f,G_m,G_f))}{V(Y_m|Y_f,G_m,G_f)}}_{=0} + \underbrace{\rho\,\kappa_f \frac{Cov(G_f,(Y_m|Y_f,G_m,G_f))}{V(Y_m|Y_f,G_m,G_f)}}_{=0} + \beta_f \underbrace{\frac{Cov(Y_f,(Y_m|Y_f,G_m,G_f)}{V(Y_m|Y_f,G_m,G_f)}}_{=0} \\
&\quad + \underbrace{\omega_m \rho_{em} \frac{Cov(G_m,(Y_m|Y_f,G_m,G_f))}{V(Y_m|Y_f,G_m,G_f)}}_{=0} + \underbrace{\omega_f \rho_{ef} \frac{Cov(G_f,(Y_m|Y_f,G_m,G_f))}{V(Y_m|Y_f,G_m,G_f)}}_{=0} \\
&\quad + \omega_m \lambda_{em} \frac{Cov(EC_m,(Y_m|Y_f,G_m,G_f))}{V(Y_m|Y_f,G_m,G_f)} + \omega_m \zeta_{em} \frac{Cov(GC_m,(Y_m|Y_f,G_m,G_f))}{V(Y_m|Y_f,G_m,G_f)} \\
&\quad + \omega_f \lambda_{ef} \frac{Cov(EC_f,(Y_m|Y_f,G_m,G_f))}{V(Y_m|Y_f,G_m,G_f)} + \omega_f \zeta_{ef} \frac{Cov(GC_f,(Y_m|Y_f,G_m,G_f))}{V(Y_m|Y_f,G_m,G_f)}
\end{aligned} \tag{41}
$$

The difference between β_m with and without controls is depicted below.

$$
\begin{aligned}
\hat{\beta}_m - \hat{\beta}_m^2 = {} & \underbrace{\underbrace{\rho\,\kappa_m \frac{Cov(G_m,(Y_m|Y_f))}{V(Y_m|Y_f)}}_{\text{Estimated NGC}} + \underbrace{\omega_m \rho_{em} \frac{Cov(G_m,(Y_m|Y_f))}{V(Y_m|Y_f)}}_{\text{Parental genetic nurture}}}_{\textbf{Broad genetic confounding}} \\
& + \underbrace{\omega_m \zeta_{em} \left(\frac{Cov(GC_m,(Y_m|Y_f))}{V(Y_m|Y_f)} - \frac{Cov(GC_m,(Y_m|Y_f,G_m,G_f))}{V(Y_m|Y_f,G_m,G_f)} \right)}_{\text{Grandparental genetic nurture mother (residual)}} \\
& \qquad \textbf{Broad genetic confounding} \\
& + \underbrace{\left(\omega_f \rho_{ef} + \rho\,\kappa_f\right) \frac{Cov(G_f,(Y_m|Y_f))}{V(Y_m|Y_f)}}_{\text{Conditional assortative mating}} \\
& + \underbrace{\omega_f \zeta_{ef} \left(\frac{Cov(GC_f,Y_m|Y_f)}{V(Y_m|Y_f)} - \frac{Cov(GC_f,(Y_m|Y_f,G_m,G_f))}{V(Y_m|Y_f,G_m,G_f)} \right)}_{\text{Cond. assort. mating + grandparental genetic nurture father (residual)}} \\
& + \underbrace{\omega_f \lambda_{ef} \left(\frac{Cov(EC_f,(Y_m|Y_f))}{V(Y_m|Y_f)} - \frac{Cov(EC_f,(Y_m|Y_f,G_m,G_f))}{V(Y_m|Y_f,G_m,G_f)} \right)}_{\text{Cond. assort. mating + environmental confounding + pop. stratification father (residual)}} \\
& + \underbrace{\omega_m \lambda_{em} \left(\frac{Cov(EC_m,(Y_m|Y_f))}{V(Y_m|Y_f)} - \frac{Cov(EC_m,(Y_m|Y_f,G_m,G_f))}{V(Y_m|Y_f,G_m,G_f)} \right)}_{\text{Environmental confounding + pop. stratification mother (residual)}}
\end{aligned} \tag{42}
$$

Relaxing our model assumptions leads to the conclusion that our baseline estimate for broad genetic confounding is similar to our baseline estimate in (14), but has a few additional terms. First, broad genetic confounding now also includes a residual term associated with genetic nurture from grandparents. This residual

term is not a bias, since it still captures indirect yet causal effects of ancestors' genes. Second, the estimate may also capture conditional assortative mating and several residual terms related to environmental confounding and population structure (i.e., the last four terms of equation 42). We discuss these terms in turn.

Residual terms The residual terms (last three terms of equation 42) are a combination of conditional assortative mating, genetic nurture, environmental confounding, and population stratification. It should be emphasized that these terms are residuals – they only arise if the conditional covariances between two characteristics (e.g., between EC_m and Y_m in the final term of equation 42) is different when conditioning on Y_f only compared with conditioning on Y_f, G_m and G_f. The first two of these residual terms stem from a possible correlation between paternal circumstances – genetically driven, GC_f, or purely environmental in origin, EC_f – and maternal educational attainment Y_m. This is again an example of conditional assortative mating, where father's characteristics correlate with maternal educational attainment, even conditional on father's educational attainment. The final term is akin to our baseline equation (9).

The ALSPAC dataset provides limited detail on the environments in which parents were raised given that its focus is on the children. As such, we attempt to assess the potential magnitude of environmental confounding by examining a set of proxy variables. These include grandparental characteristics that likely shaped parental environments (e.g., maternal grandparents' years of education, or whether they are involved in providing childcare), as well as parental outcomes that may partly reflect early-life exposures (e.g., maternal social class and birth weight). Table F3 presents the estimated covariance-to-variance ratios between these proxies and residual parental education, comparing specifications with and without genetic controls.

Table F3 shows the resulting coefficient estimates for a list of environmental circumstances. Across all variables considered, the difference between the two specifications is minimal, suggesting that the magnitude of these residual terms is likely to be very small. For instance, regressing grandmother's years of education on father's years of education conditional on mother's years of education produces a coefficient of 0.107. Additionally controlling for both parent's PGIs changes the coefficient slightly to 0.092. Hence the term within parentheses (denoted by Δ in the Table) in this case is 0.015. Overall, it seems plausible that the magnitude of these residual environmental terms is very small.

	$\frac{Cov(C_m,\tilde{Y}_m)}{V(\tilde{Y}_m)}$	$\frac{Cov(C_m,\tilde{Y}_m^{gc})}{V(\tilde{Y}_m^{gc})}$	$[\Delta]$	$\frac{Cov(C_m,\tilde{Y}_f)}{V(\tilde{Y}_f)}$	$\frac{Cov(C_m,\tilde{Y}_f^{gc})}{V(\tilde{Y}_f^{gc})}$	$[\Delta]$
Social class (mom)	-0.219	-0.205	-0.014	-0.078	-0.070	-0.008
Social class (father)	-0.058	-0.041	-0.018	-0.338	-0.330	-0.009
Childcare by grandparents	-0.031	-0.026	-0.005	-0.032	-0.030	-0.003
Grandfather educ. (mom, yrs)	0.198	0.156	0.042	0.115	0.095	0.020
Grandmother educ. (mom, yrs)	0.158	0.120	0.038	0.107	0.092	0.015
Birthweight (mom, kgs)	0.006	-0.001	0.007	0.005	0.003	0.002
Grandfather involved (mom)	-0.010	-0.007	-0.003	-0.013	-0.012	-0.002
Grandmother involved (mom)	-0.016	-0.015	-0.002	-0.019	-0.017	-0.002

Table F3 Estimates of covariance to variance ratios between parental environment proxies and residual parental education. Residuals are obtained from regressions controlling for the other parent's education, with and without controls for parental polygenic indices. Social class is a variable coded based on parental occupation, where 1 indicates professional occupations, 2 managerial and technical, 3 skilled (non-manual or manual), 4 partly skilled, and 5 unskilled. Childcare by grandparents is a binary variable equal to 1 if either grandparent provides any childcare. Grandfather's education and grandmother's education refer to the years of schooling completed by the maternal grandparents. Birth weight corresponds to the mother's own birth weight in kilograms. Grandfather involved and grandmother involved are binary indicators equal to 0 if the respective maternal grandparent is involved in activities with the grandchild.

The final two residual terms partially involve population stratification. In Appendix G.2 we add the 20 first principal components of the mother and child, and our results remain virtually unchanged. Of course, residual population stratification can still occur when there are geographic or regional differences in allele frequencies relating to a trait of interest that cannot necessarily be controlled for with principal components (e.g., Haworth et al., 2019). Still, ALSPAC is known to be a very homogeneous genetic group, as it focuses on white ancestry individuals in a small geographic region of the UK (Fraser et al., 2013). This is reflected is a high genetic homogeneity (Ruisch et al., 2019). In this sense, it is plausible that our estimates are less affected by bias from residual population stratification than other datasets, with more heterogeneous populations.

Conditional Assortative mating The term "conditional assortative mating" in equation (42) directly follows from relaxing assumption A.2. If assortative mating does not just take place at the level of educational attainment, then the father's genetic endowments may still correlate with mother's educational attainment conditional on father's educational attainment. We can partially test our assumption that assortative mating occurs exclusively at the educational attainment level. To do this, we compute the terms $\frac{Cov(G_f,(Y_m|Y_f))}{V(Y_m|Y_f)}$ and $\frac{Cov(G_m,(Y_f|Y_m))}{V(Y_f|Y_m)}$. Table F4 shows that controlling for the partner's educational attainment controls for a large share of assortative mating; 49% for mothers and 62% for fathers. This suggests that educational attainment is the most important, albeit not unique, sorting mechanism, as the association remains statistically significant, indicating that other traits also contribute to assortative mating.

	EA PGI father (UKB)		EA PGI mother (UKB)	
	(1)	(2)	(3)	(4)
Mother years of education	0.120***	0.061***		0.179***
	(0.009)	(0.010)		(0.010)
Father years of education		0.104***	0.146***	0.056***
		(0.010)	(0.008)	(0.009)
R-squared	0.043	0.070	0.072	0.142
N	4,031	4,031	4,031	4,031

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Table F4 Results of the OLS regressions explaining the maternal and paternal EA PGI. Robust standard errors in parenthesis.

To gauge the magnitude of the unaccounted assortative mating, we would like to know how large the term $\left(\omega_f \rho_{ef} + \rho \kappa_f\right) \frac{Cov(G_f,(Y_m|Y_f))}{V(Y_m|Y_f)}$ is. While we don't know $\omega_f \rho_{ef}$ we can calculate $\rho \kappa_f \frac{Cov(G_f,(Y_m|Y_f))}{V(Y_m|Y_f)}$ and $\rho \kappa_m \frac{Cov(G_m,(Y_f|Y_m))}{V(Y_f|Y_m)}$ for fathers, where G_m and G_f are corrected for measurement error using the technique described in Appendix E. The terms are equal to 39.77*0.50*0.09=1.78 for mothers and 39.77*0.50*0.08=1.59 fathers. We can deduct this term to our broad genetic confounding estimates of Table 2: for mothers, broad genetic confounding is 6.42-1.78=4.64, and for fathers 5.11-1.59=3.52. Accounting for this additional part of broad genetic confounding reduces our estimates of broad genetic confounding from 45.1% to 32.6% for mothers and from 41.1% to 28.5% for fathers. These differences are upper bounds of the broad genetic confounding estimate as one would still need to deduct $\omega_f \rho_{ef} \frac{Cov(G_f,(Y_m|Y_f))}{V(Y_m|Y_f)}$, which is likely a positive term.

An alternative approach to overcome the bias induced by conditional assortative mating is to control for the partner's genetic endowments in the baseline equation. This approach starts from a different baseline estimator that also controls for G_f in addition to Y_f. Let's call the resulting estimator $\hat{\beta}_m^G$, where the G superscript denotes

that we additionally controlled for G_f:

$$
\begin{aligned}
\hat{\beta}_m^G &= \frac{Cov(Y_c,(Y_m|Y_f,G_f))}{V(Y_m|Y_f,G_f)} \\
&= \beta_m + \rho\frac{Cov(G_c,(Y_m|Y_f,G_f))}{V(Y_m|Y_f,G_f)} + \omega_m\frac{Cov(E_m,(Y_m|Y_f,G_f))}{V(Y_m|Y_f,G_f)} + \omega_f\frac{Cov(E_f,(Y_m|Y_f,G_f))}{V(Y_m|Y_f,G_f)} \\
&= \beta_m + \underbrace{\rho\kappa_m\frac{Cov(G_m,(Y_m|Y_f,G_f))}{V(Y_m|Y_f,G_f)}}_{\text{Estimated NGC (biased down)}} + \underbrace{\omega_m\rho_{em}\frac{Cov(G_m,(Y_m|Y_f,G_f))}{V(Y_m|Y_f,G_f)}}_{\text{Parental Genetic Nurture (biased down)}} + \underbrace{\omega_m\zeta_{em}\frac{Cov(GC_m,(Y_m|Y_f,G_f))}{V(Y_m|Y_f,G_f)}}_{\text{Grandparental Genetic Nurture (biased down)}} \\
&\quad + \underbrace{\omega_m\lambda_{em}\frac{Cov(EC_m,(Y_m|Y_f,G_f))}{V(Y_m|Y_f,G_f)}}_{\text{Environmental confounding + population stratification}} \\
&\quad + \underbrace{\underbrace{\left(\omega_f\rho_{ef}+\rho\kappa_f\right)\frac{Cov(G_f,(Y_m|Y_f,G_f))}{V(Y_m|Y_f,G_f)}}_{=0} + \omega_f\lambda_{ef}\frac{Cov(EC_f,(Y_m|Y_f,G_f))}{V(Y_m|Y_f,G_f)} + \omega_f\zeta_{ef}\frac{Cov(GC_f,(Y_m|Y_f,G_f))}{V(Y_m|Y_f,G_f)}}_{\text{Conditional Assortative Mating}}
\end{aligned}
\tag{43}
$$

$$
\begin{aligned}
\hat{\beta}_m^G - \hat{\beta}_m^2 = &\underbrace{\underbrace{\rho\kappa_m\frac{Cov(G_m,(Y_m|Y_f,G_f))}{V(Y_m|Y_f,G_f)}}_{\text{Estimated NGC (BD)}} + \underbrace{\omega_m\rho_{em}\frac{Cov(G_m,(Y_m|Y_f))}{V(Y_m|Y_f)}}_{\text{Parental genetic nurture (BD)}}}_{\textbf{Broad genetic confounding (BD)}} \\
&+ \underbrace{\omega_m\zeta_{em}\left(\frac{Cov(GC_m,(Y_m|Y_f,G_f))}{V(Y_m|Y_f,G_f)} - \frac{Cov(GC_m,(Y_m|Y_f,G_m,G_f))}{V(Y_m|Y_f,G_m,G_f)}\right)}_{\text{Grandparental genetic nurture mother (residual, BD)}} \\
&\quad \textbf{Broad genetic confounding (BD)} \\
&+ \underbrace{\omega_f\zeta_{ef}\left(\frac{Cov(GC_f,Y_m|Y_f,G_f)}{V(Y_m|Y_f,G_f)} - \frac{Cov(GC_f,(Y_m|Y_f,G_m,G_f))}{V(Y_m|Y_f,G_m,G_f)}\right)}_{\text{Cond. assort. mating + grandparental genetic nurture father (residual)}} \\
&+ \underbrace{\omega_f\lambda_{ef}\left(\frac{Cov(EC_f,(Y_m|Y_f,G_f))}{V(Y_m|Y_f,G_f)} - \frac{Cov(EC_f,(Y_m|Y_f,G_m,G_f))}{V(Y_m|Y_f,G_m,G_f)}\right)}_{\text{Cond. assort. mating + environmental confounding + pop. stratification father (residual)}} \\
&+ \underbrace{\omega_m\lambda_{em}\left(\frac{Cov(EC_m,(Y_m|Y_f,G_f))}{V(Y_m|Y_f,G_f)} - \frac{Cov(EC_m,(Y_m|Y_f,G_m,G_f))}{V(Y_m|Y_f,G_m,G_f)}\right)}_{\text{Environmental confounding + pop. stratification mother (residual)}}
\end{aligned}
\tag{44}
$$

Ignoring the residual terms, this difference simplifies to

$$
\hat{\beta}_m^G - \hat{\beta}_m^2 \approx \left(\rho\kappa_m + \omega_m\zeta_{em}\right)\frac{Cov(G_m,(Y_m|Y_f,G_f))}{V(Y_m|Y_f,G_f)} \tag{45}
$$

The advantage of this estimation is that it gets rid of the residual assortative mating term and only captures broad genetic confounding. However, the estimate of BGC may be slightly underestimated, given that the father's genes will correlate with the mothers and the child's. As such, we know that:

$$\frac{Cov(G_m,(Y_m|Y_f,G_f))}{V(Y_m|Y_f,G_f)} < \frac{Cov(G_m,(Y_m|Y_f))}{V(Y_m|Y_f)}$$

To get a feeling for the size of the bias, we add the other parent's EA PGI as a control in our baseline specification. Table F5 implements this estimate, where the partner's EA PGI is always added as control. Due to this, our ORIV estimates of broad genetic confounding reduce from 6.4 to 4.42 for mothers and 5.1 to 3.6 for fathers. This leads to the conclusion that the percentage of the parent-child association explained by broad genetic confounding is likely leaning towards 31.1% for mothers and 28.9% for fathers. This fits well with our previous estimate, of 32.6% and 28.5%.

	OLS			ORIV		
	(1)	(2)	(3)	(4)	(5)	(6)
Mother years of education	13.007***	11.465***	10.460***	12.219***	9.565***	7.797***
	(1.298)	(1.358)	(1.354)	(0.941)	(1.054)	(1.072)
Father years of education	10.324***	11.459***	9.568***	8.627***	10.789***	7.224***
	(1.250)	(1.245)	(1.249)	(0.934)	(0.895)	(0.949)
EA PGI father	19.416***		18.765***	26.986***		26.397***
	(2.031)		(2.006)	(2.181)		(2.176)
EA PGI mother		15.454***	14.543***		21.522***	20.678***
		(2.113)	(2.074)		(2.282)	(2.284)
R-squared	0.117	0.109	0.127	0.091	0.094	0.088
N	4032	4032	4032	8064	8064	8064
Broad genetic conf mom	.	.	2.547	.	.	4.423
Broad genetic conf dad	.	.	1.891	.	.	3.565

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Table F5 Results of the OLS regressions explaining the maternal and paternal EA PGI. Robust standard errors in parenthesis.

In conclusion, relaxing the assumptions of our model reveals that our broad genetic confounding estimates may be subject to an upward bias arising mostly from assortative mating based on other characteristics besides education. Compared to adoption and twin approaches, a key advantage of using molecular genetic data is its ability to quantify and account for genetic sources of assortative mating.

G Results of robustness checks

G.1 Results with other measures of Key Stage 4 grades

This section replicates the main results but with alternative measures of the Key Stage 4 results. While our baseline measure aggregates all points across examinations, here we employ a measure of average point per exam, and the highest point score obtain in the Key Stage 4 Science exam. Table G6 shows the summary statistics of these two measures. The maximum number of points decreases from 1,171 (see Table 1) to 61 and 58, respectively.

	Mean	S.D.	Min.	Max.	*N*
Key stage 4 average points per exam	41.29	8.76	0	61	4,030
Key stage 4 highest point score in the Science exam	43.47	9.39	0	58	3,861

Table G6 Summary statistics of alternative Key Stage 4 measures (ALSPAC). S.D.=Standard deviation; Min.=Minimum; Max.=Maximum

Table G7 and G8 replicate our baseline results depicted in Table 2, but with average Key Stage 4 point per exam and highest point score obtained in the Science exam. The percentages of broad and narrow genetic confounding are strikingly similar to the ones obtained in our baseline estimation. If anything, genetic confounding for fathers seems to be slightly lower for the highest points obtained in the Science exam, but differences are small.

	OLS				ORIV		
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Mother years of education	1.205***		0.884***			0.662***	
	(0.080)		(0.081)			(0.065)	
Father years of education	1.098***		0.869***			0.686***	
	(0.077)		(0.077)			(0.060)	
EA PGI mother (UKB)		2.196***	1.278***	1.320***	2.654***	1.810***	1.109***
		(0.128)	(0.130)	(0.185)	(0.134)	(0.145)	(0.211)
EA PGI father (UKB)		2.114***	1.514***	0.880***	2.624***	2.054***	0.471
		(0.128)	(0.127)	(0.224)	(0.139)	(0.140)	(0.269)
EA PGI child (UKB)				1.801***			3.153***
				(0.272)			(0.335)
Broad genetic conf mom			0.321			0.543	
(in %)			(26.7%)			(45.1%)	
Broad genetic conf dad			0.229			0.412	
(in %)			(20.9%)			(37.5%)	
Narrow genetic conf mom				0.160			0.387
(in %)				(13.3%)			(32.1%)
Narrow genetic conf dad				0.093			0.232
(in %)				(8.5%)			(21.1%)
R-squared	0.170	0.137	0.218	0.146	-	-	-
N	4,030	4,030	4,030	4,030	4,030	4,030	4,030

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Table G7 Results of the OLS and ORIV regressions explaining the offspring Key Stage 4 average grade per exam. Robust standard errors in parentheses.

	OLS				ORIV		
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Mother years of education	1.301***		0.968***			0.727***	
	(0.085)		(0.085)			(0.070)	
Father years of education	1.164***		0.926***			0.728***	
	(0.085)		(0.084)			(0.066)	
EA PGI mother (UKB)		2.278***	1.268***	1.347***	2.775***	1.849***	1.169***
		(0.140)	(0.143)	(0.211)	(0.150)	(0.164)	(0.238)
EA PGI father (UKB)		2.347***	1.714***	1.034***	2.930***	2.323***	0.677*
		(0.136)	(0.136)	(0.257)	(0.153)	(0.154)	(0.309)
EA PGI child (UKB)				1.919***			3.292***
				(0.313)			(0.381)
Broad genetic conf mom			0.333			0.574	
(in %)			(25.6%)			(44.1%)	
Broad genetic conf dad			0.238			0.437	
(in %)			(20.4%)			(37.5%)	
Narrow genetic conf mom				0.173			0.410
(in %)				(13.3%)			(31.5%)
Narrow genetic conf dad				0.095			0.232
(in %)				(8.2%)			(19.9%)
R-squared	0.172	0.137	0.220	0.146	-	-	-
N	3,861	3,861	3,861	3,861	3,861	3,861	3,861

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Table G8 Results of the OLS and ORIV regressions explaining the offspring Key Stage 4 grade in the Science exam. Robust standard errors in parentheses.

G.2 Results with control variables

This section replicates the main results while adding gender and the first 20 principal components of the genomic relationship matrix of the child and the mother. Adding these controls does not alter our results in any meaningful way.

	OLS				ORIV		
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Mother years of education	14.092***		10.382***			7.658***	
	(1.293)		(1.340)			(1.066)	
Father years of education	12.205***		9.476***			7.117***	
	(1.234)		(1.236)			(0.939)	
EA PGI mother		24.753***	14.365***	14.512***	30.066***	20.734***	10.209**
		(1.956)	(2.054)	(2.873)	(2.047)	(2.297)	(3.222)
EA PGI father		25.749***	18.965***	11.300**	32.990***	26.771***	5.281
		(2.002)	(2.027)	(3.577)	(2.128)	(2.200)	(4.146)
EA PGI child				20.997***			40.361***
				(4.287)			(5.115)
Female	42.205***	41.924***	42.029***	42.012***	42.346***	42.292***	42.466***
	(4.026)	(4.060)	(3.960)	(4.050)	(2.964)	(2.858)	(2.973)
Child's PCs	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Mother's PCs	Yes	Yes	Yes	Yes	Yes	Yes	Yes
R-squared	0.128	0.113	0.158	0.118	-	-	-
N	3,980	3,980	3,980	3,980	3,980	3,980	3,980

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Table G9 Results of the OLS and ORIV regressions explaining the offspring Key Stage 4 grade. Robust standard errors in parenthesis.

G.3 Results by gender

This section replicates the results separately by females and males. Table G10 depicts the results for females (daughters) and Table G11 depicts the results for males (sons). While father-son education association seems to be lower – with or without controlling for genetic transmission – the differences are small in magnitude.

	OLS				ORIV		
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Mother years of education	13.132***		9.832***			7.401***	
	(1.722)		(1.777)			(1.418)	
Father years of education	13.581***		10.591***			8.580***	
	(1.652)		(1.670)			(1.269)	
EA PGI mother		25.689***	15.014***	13.247***	30.754***	20.507***	10.687*
		(2.678)	(2.767)	(3.846)	(2.742)	(3.068)	(4.519)
EA PGI father		24.240***	17.611***	6.852	29.971***	23.611***	2.256
		(2.542)	(2.585)	(4.499)	(2.668)	(2.735)	(5.569)
EA PGI child				25.267***			39.679***
				(5.498)			(6.967)
R-squared	0.105	0.085	0.136	0.093	-	-	-
N	2,104	2,104	2,104	2,104	2,104	2,104	2,104

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Table G10 Results of the OLS and ORIV regressions explaining the offspring Key Stage 4 grade, for the subsample of daughters. Robust standard errors in parenthesis.

	OLS				ORIV		
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Mother years of education	14.821***		10.607***			7.538***	
	(1.897)		(1.986)			(1.571)	
Father years of education	11.558***		9.054***			6.350***	
	(1.807)		(1.798)			(1.368)	
EA PGI mother (UKB)		24.087***	13.715***	15.756***	29.207***	20.736***	10.095*
		(2.797)	(2.976)	(4.133)	(2.983)	(3.327)	(4.453)
EA PGI father (UKB)		27.308***	20.147***	15.471**	36.192***	29.833***	9.348
		(2.999)	(3.040)	(5.319)	(3.276)	(3.421)	(5.902)
EA PGI child (UKB)				17.381**			40.253***
				(6.342)			(7.222)
R-squared	0.095	0.082	0.125	0.085	-	-	-
N	1,928	1,928	1,928	1,928	1,928	1,928	1,928

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Table G11 Results of the OLS and ORIV regressions explaining the offspring Key Stage 4 grade, for the subsample of sons. Robust standard errors in parenthesis.

G.4 Results by education level

This section uses parental education levels instead of parental years of education to estimate broad genetic confounding. In this section we exclude the only family whose mother reported having no educational degree. Figure 6 depicts the distribution of parental educational attainment. Table G12 replicates the main results with a binary variable for each education level and having a CSE as baseline. The results reveal that the raw association between parental and offspring education is roughly linear over the additional degrees. The results also reveal larger attenuation percentages for higher levels of education. For mothers, the coefficient reduces 30.5% for having a degree (from 109.1 to 75.8), for A-levels 23.3% (from 74.9 to 57.6), for O-levels 15.8% (from 52.7 to 44.4), and vocational 10.4% (from 27.1 to 24.3). For fathers, the coefficient for having a degree reduces 34.3% (from 75.3 to 49.5), for A-levels 24.8% (from 45.3 to 34.1), for O-levels 18-8% (from 45.5 to 34.1), and for vocational 6.72% (from 10.6 to 9.9 albeit the coefficients are not significant). This suggests that broad genetic confounding plays a larger role in the transmission of higher levels of education.

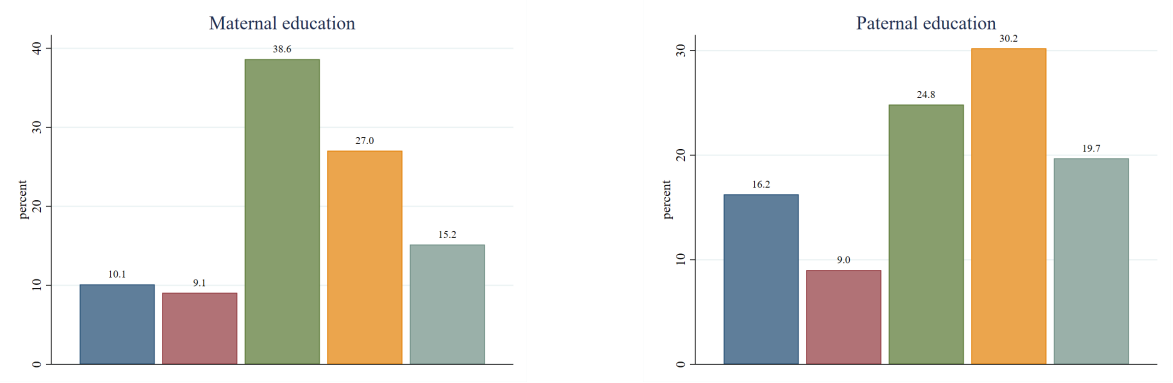

Figure 6 Distribution of Maternal and Paternal education. The blue bar corresponds to having CSE or O-levels, the red column to having a vocational education, the green to having A-levels, and the yellow to having a university degree.

	OLS			ORIV	
	(1)	(2)	(3)	(4)	(5)
Mother education: Vocational	27.103**		24.065*		24.286***
	(10.174)		(10.051)		(7.169)
Mother education: O level	52.715***		47.627***		44.391***
	(7.874)		(7.829)		(5.669)
Mother education: A level	74.875***		64.370***		57.462***
	(8.264)		(8.312)		(6.197)
Mother education: Degree	109.069***		89.983***		75.768***
	(9.569)		(9.845)		(7.675)
Father education: Vocational	10.642		11.161		9.927
	(8.474)		(8.408)		(6.040)
Father education: O level	33.496***		29.839***		27.198***
	(6.679)		(6.584)		(4.741)
Father education: A level	45.319***		39.604***		34.093***
	(6.558)		(6.481)		(4.709)
Father education: Degree	75.279***		61.304***		49.486***
	(7.789)		(7.795)		(5.841)
EA PGI mother		25.014***	11.807***	41.516***	23.593***
		(1.977)	(2.094)	(2.812)	(3.241)
EA PGI father		25.523***	17.382***	46.425***	35.580***
		(1.980)	(1.984)	(2.994)	(3.074)
R-squared	0.127	0.081	0.150	-	-
N	4,031	4,031	4,031	4,031	4,031

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Table G12 Results of the OLS and ORIV regressions explaining the offspring Key Stage 4 grade. Maternal and paternal education is a categorical variable that ranges from having CSE or O-levels, having a vocational education, having A-levels, or having a university degree. Robust standard errors in parenthesis.

G.5 Robustness of the ORIV estimates

Table G13 presents the results of the first stage regressions predicting each PGI. Each independent PGI is highly predictive of the other (that is, the child's PGI based on 23andMe is highly predictive of the child's PGI based on UKB, and vice versa). The F-statistic varies between approximately 382 and 1542.

	EA PGI (UKB)				
	child	mother		father	
	(1)	(2)	(3)	(4)	(5)
EA PGI child (23andMe)	0.513***				
	(0.014)				
EA PGI mother (23andMe)		0.525***	0.453***		-0.009
		(0.013)	(0.013)		(0.014)
EA PGI father (23andMe)			0.022	0.494***	0.458***
			(0.013)	(0.014)	(0.014)
Mother years of education			0.116***		0.044***
			(0.009)		(0.009)
Father years of education			0.033***		0.063***
			(0.008)		(0.009)
F-statistic	1388.569	1542.574	497.194	1294.959	382.308
R-squared	0.263	0.276	0.331	0.244	0.271
N	4,032	4,032	4,032	4,032	4,032

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Table G13 Results of the OLS (first stage) predicting the the EA PGI based on the summary statistics of the UK Biobank data set. Robust standard errors in parenthesis.

Table G14 presents our IV results with the 23andMe-based PGI instrumenting for the UKB-based PGI (columns 1, 3, 5, and 7), and vice versa (columns 2, 4, 6, 8). The point estimates differ somewhat, illustrating the benefit of ORIV rather than arbitrarily relying on one of the two sets of estimates.

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Mother years of education					12.012***	12.493***	9.377***	9.896***
					(1.331)	(1.331)	(1.525)	(1.459)
Father years of education					8.668***	8.640***	10.795***	10.819***
					(1.317)	(1.324)	(1.262)	(1.269)
EA PGI father (UKB)	48.825***				35.413***			
	(4.095)				(4.293)			
EA PGI father (23andMe)		58.561***				41.263***		
		(4.330)				(4.466)		
EA PGI mother (UKB)			44.083***				27.190***	
			(3.910)				(4.474)	
EA PGI mother (23andMe)				54.257***				31.951***
				(3.963)				(4.396)
N	4,032	4,032	4,032	4,032	4,032	4,032	4,032	4,032

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Table G14 Results of the Two Stage Least Square (2SLS) predicting the Key Stage 4 grade. The father's (mother's) UKB PGI is used as an instrument for the father's (mother's) 23andME PGI and vice-versa. Robust standard errors in parenthesis.

Finally, we replicate the main results with two independent PGIs built from two non-overlapping samples of the UK Biobank. Given that the discovery sample is based on individuals residing in the same country, the discovery sample is expected to be more homogeneous and the PGIs are expected to have a larger correlation with each other. Figure 7 depicts the relationship between the Key Stage 4 of the child and the PGIs. One can observe a clear positive relationship between the grade and the PGIs. Since these PGIs were constructed based on smaller discovery samples, their predictive power is smaller than the PGIs constructed using the full UK Biobank and 23andMe as discovery samples.

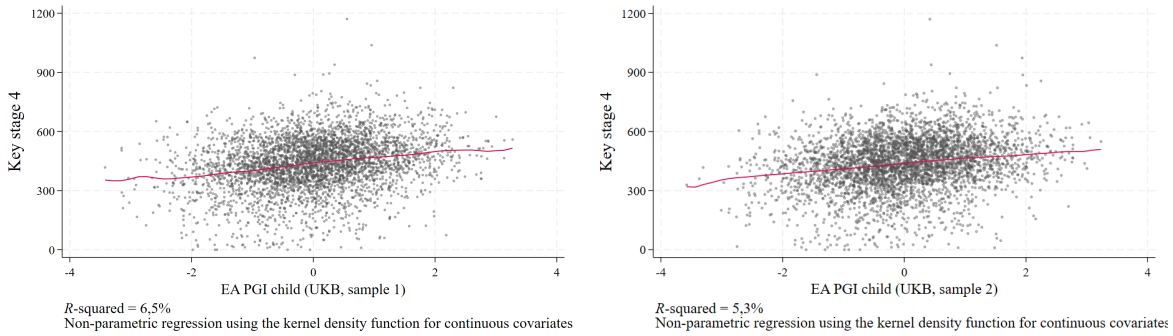

Figure 7 Scatterplot of Key Stage 4 and the EA PGI of the child with a locally weighted regression line. On the left, the PGI based on the summary statistics of one half of the UK Biobank sample. On the right, the PGI based on the summary statistics of the other half of the UK Biobank sample.

Table G15 replicates the results of Table 2 but with the split sample PGIs. Comparing columns 1 and 5 one can conclude that the coefficients of maternal and paternal education shrink by approximately 35% each, slightly less than in our main specification. This can be explained by the lower predictive power of these PGIs. Qualitatively, all the results from the original specification hold.

To dig a little deeper into this, Table G16 shows the relationship between the two independent EA PGIs for the child, mother, and father. While in theory, the correlation between the PGIs could be larger under this specification since the PGIs originate from the same discovery sample, this doesn't hold in practice, perhaps due to the lower predictive sample of these PGIs. While under the original specification the coefficients of a regression explaining the child, mother, and father UKB PGI with the other 23andMe PGI is 0.513, 0.525, and 0.494 – see Table G13, –, here the coefficients of the same regressions are 0.507, 0.497, and 0.464 - Table G16. Likewise, the F-statistics are slightly smaller – 1377.1 versus 1388.6, 1542.6 versus 1331.7, and 1295.0 versus 1081.9. In sum, it seems that the statistical properties of the two-sample ORIV estimates are slightly better, and since ORIV is robust against small deviations from a genetic correlation of 1 (see van Kippersluis et al., 2023), the two-sample ORIV estimates constitutes our main estimates.

	OLS				ORIV		
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Mother years of education	14.214***		11.470***			9.160***	
	(1.305)		(1.340)			(1.061)	
Father years of education	12.334***		10.135***			8.067***	
	(1.246)		(1.248)			(0.942)	
EA PGI mother		22.385***	12.876***	13.716***	26.098***	15.895***	12.360***
		(2.027)	(2.056)	(2.890)	(2.157)	(2.347)	(3.232)
EA PGI father		23.766***	17.009***	11.194**	30.687***	24.353***	10.530*
		(2.025)	(2.007)	(3.592)	(2.266)	(2.276)	(4.245)
EA PGI child				18.023***			29.573***
				(4.266)			(5.213)
R-squared	0.097	0.066	0.122	0.070	-	-	-
N	4,032	4,032	4,032	4,032	4,032	4,032	4,032

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Table G15 Results of the OLS and ORIV regressions explaining the offspring Key Stage 4 grade. Robust standard error in parenthesis.

	EA PGI (UKB, sample 1)		
	child	mother	father
	(1)	(2)	(3)
EA PGI child (UKB, sample 2)	0.507***		
	(0.014)		
EA PGI mother (UKB, sample 2)		0.497***	
		(0.014)	
EA PGI father (UKB, sample 2)			0.464***
			(0.014)
F-statistic	1377.115	1331.788	1081.895
R-squared	0.258	0.247	0.216
N	4,032	4,032	4,032

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Table G16 Results of the OLS regression predicting each sample 2 EA PGI based on the summary statistics of sample 1 of the UK Biobank data set. Robust standard errors in parenthesis.

G.6 Results using trios with non-imputed genetic information

This section replicates the main analysis on the subsample of trios for which there is actual genetic imputation (see Appendix D). For the 1,022 trios for which we have genetic information, we imputed the father genomes, such that we could compare the imputed and real paternal PGIs. Table G17 depicts pairwise correlations between imputed and actual PGIs for the fathers. The imputed and actual PGIs constructed using the same summary statistics have a correlation of 0.66-0.67, indicating a relatively large accuracy in predicting the paternal genome based on the mother and the child's.

	EA PGI father (23andMe, non-imputed)	EA PGI father (23andMe, imputed)	EA PGI father (UKB, non-imputed)	EA PGI father (UKB, imputed)
EA PGI father (23andMe, non-imputed)	1.00			
EA PGI father (23andMe, imputed)	0.67***	1.00		
EA PGI father (UKB, non-imputed)	0.56***	0.37***	1.00	
EA PGI father (UKB, imputed)	0.36***	0.46***	0.66***	1.00

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Table G17 Pairwise correlations between imputed and non-imputed EA PGI for fathers. N=1,022.

Table G18 replicates the main results on the reduced sample of genotyped trios. In this subsample, broad genetic confounding is estimated to be 24.2-31.4% and narrow genetic confounding is 19.7-31.0%. Broad genetic confounding is estimated to be lower than the attenuation in our original specification, and differences between mothers and fathers are estimated to be slightly larger. However, since the raw intergenerational association is also different compared to our main sample, in particular for fathers, it suggests that the samples are also simply somewhat different. Table G19 replicates the first stage regressions for this subsample, for both observed and imputed parental PGI's. It is evident that regardless of the PGI's utilized, the first stage is strong and the F-statistic is well above 10. Overall, this specification is qualitatively similar to our main specification.

	OLS				ORIV		
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Mother years of education	13.983***		12.439***			10.605***	
	(2.308)		(2.370)			(1.819)	
Father years of education	7.279**		6.471**			4.995**	
	(2.276)		(2.375)			(1.764)	
EA PGI mother		19.499***	7.777*	15.443***	25.151***	13.234**	14.336**
		(3.788)	(3.811)	(4.504)	(3.975)	(4.390)	(4.926)
EA PGI father		11.515**	2.694	7.763	15.047***	6.929	4.177
		(3.591)	(3.663)	(4.450)	(3.734)	(3.914)	(5.037)
EA PGI child				8.087			22.007***
				(5.298)			(6.529)
Broad genetic conf. mother			1.544			3.378	
Broad genetic conf. father			0.807			2.284	
Narrow genetic conf mom	.	.	.	0.729	.	.	2.748
Narrow genetic conf dad	.	.	.	0.620	.	.	2.256
R-squared	0.094	0.041	0.098	0.043	-	-	-
N	1,022	1,022	1,022	1,022	1,022	1,022	1,022

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Table G18 Results of the OLS and ORIV regressions explaining the offspring Key Stage 4 grade, for the subsample of genotyped trios. Robust standard errors in parenthesis. All PGI's included are derived from observed genetic data.

	EA PGI (UKB)		
	child	mother	father
	(1)	(2)	(3)
EA PGI child (23andMe, observed)	0.503***		
	(0.027)		
EA PGI mother (23andMe, observed)		0.522***	
		(0.026)	
EA PGI father (23andMe, observed)			0.559***
			(0.026)
F-statistic	342.124	394.623	479.974
R-squared	0.253	0.272	0.312
N	1,022	1,022	1,022

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Table G19 Results of the OLS regression predicting each EA PGI based on the summary statistics of the UK Biobank data set, for the subsample of genotyped trios. Robust standard errors in parenthesis.

G.7 Results with an interaction term

This section extends the main OLS results by including interaction terms. Table G20, columns 1 and 2 include interactions between maternal and paternal education and each EA PGI. We find that the coefficients are close to zero and insignificant in all cases, except a marginally significant negative interaction between father years of education and father's EA PGI. Table G20, columns 3 and 4, report interactions between maternal and paternal education and between maternal and paternal PGIs. We find evidence of non-linearities in the case of the parental PGIs: the returns to a parent's EA PGI decline as the partner's EA PGI increases. This suggests that having at least one parent with a high EA PGI is particularly important for educational success. By contrast, the interaction at the education level is small in magnitude and statistically insignificant, indicating no evidence of non-linearities in parental education. Accounting for this interaction leads to broad genetic confounding estimates of 5.0 (21.7%) for mothers and 4.0 (19%) for fathers, similar to our OLS baseline estimates of 26.4% and 22.4%, respectively.

	(1)	(2)	(3)	(4)
Mother years of education	11.480***	11.276***	23.172**	18.140*
	(1.362)	(1.392)	(8.187)	(8.128)
Father years of education	10.098***	10.201***	20.903**	16.919*
	(1.265)	(1.272)	(7.892)	(7.827)
EA PGI child (UKB)	68.808***			
	(15.987)			
EA PGI child (UKB)	0.000			
	(.)			
EA PGI mother (UKB)	3.147	37.267**		14.447***
	(2.917)	(13.641)		(2.071)
EA PGI father (UKB)	2.681	50.228***		18.648***
	(3.469)	(13.347)		(1.994)
Mother years of education × EA PGI child (UKB)	-2.335			
	(1.207)			
Father years of education × EA PGI child (UKB)	-1.314			
	(1.153)			
Mother years of education × EA PGI mother (UKB)		-1.831		
		(1.070)		
Father years of education × EA PGI father (UKB)		-2.485*		
		(1.016)		
Mother years of education × Father years of education			-0.662	-0.555
			(0.595)	(0.592)
EA PGI mother (UKB) × EA PGI father (UKB)				-5.171**
				(1.866)
R-squared	0.135	0.129	0.097	0.129
N	4032	4032	4032	4032

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Table G20 Results of the OLS regressions explaining the offspring Key Stage 4 grade. Robust standard errors in parenthesis.

G.8 Results without accounting for assortative mating

This section replicates the results by analyzing the mother-child or father-child association separately. Given assortative mating, meaning that couples tend to be more similar to each other than any two random individuals, it is likely that including a single parent will lead to an upward bias on the coefficient of parental educational attainment. Table G21 confirms this prediction for mother-child pairs and Table G22 does the same for father-child pairs. Indeed, including only one parent educational attainment results in an upward bias of the estimates, both before and after controls for genetic transmission are employed. While OLS without controls finds an association of 21.0 for mothers and 19.4 for fathers – column 1, G21 and G22 – our original estimation finds and association of 14.2 for mothers and 12.3 for fathers. In our main specification, where we employ an ORIV estimation that controls for genetic confounding, we find an association of 14.9 for mothers and 14.35 for fathers – column 8 of Tables G21 and G22. This leads to a broad confounding estimate of 29% for mothers and 26% for fathers, much lower than in our baseline specification of 41-45%. The reasoning for this downward bias is that the maternal (paternal) education highly associates with paternal (maternal) education. In contrast the association between education PGI's of parents is lower. As such, maternal (paternal) education will pick up a larger chunk of the partner education than the maternal (paternal) PGI is picking up of the partner's genetic advantage. As such, only considering one of the parents introduces a mechanical bias in the genetic confounding estimates.

	OLS				ORIV		
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Mother years of education	21.049***		17.430***			14.935***	
	(1.098)		(1.171)			(0.958)	
EA PGI mother		28.492***	17.297***	9.807***	35.631***	23.906***	8.115**
		(2.026)	(2.120)	(2.357)	(2.014)	(2.283)	(2.577)
EA PGI child				32.393***			45.541***
				(2.386)			(2.620)
R-squared	0.076	0.045	0.091	0.085	-	-	-
N	4,032	4,032	4,032	4,032	4,032	4,032	4,032

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Table G21 Results of the OLS and ORIV regressions explaining the offspring Key Stage 4 grade. Robust standard errors in parenthesis.

	OLS				ORIV		
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Father years of education	19.439***		16.568***			14.346***	
	(1.062)		(1.083)			(0.842)	
EA PGI father (UKB)		28.918***	21.329***	0.925	37.731***	29.222***	-2.084
		(2.020)	(2.047)	(2.902)	(2.092)	(2.190)	(3.309)
EA PGI child (UKB)				37.357***			52.038***
				(2.942)			(3.269)
R-squared	0.072	0.047	0.096	0.081	-	-	-
N	4,032	4,032	4,032	4,032	4,032	4,032	4,032

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Table G22 Results of the OLS and ORIV regressions explaining the offspring Key Stage 4 grade. Robust standard errors in parenthesis.

G.9 Results with standardized variables

Table G23 presents the results of the OLS and ORIV regressions predicting Key Stage 4 grade, where all variables are standardized. The implied *R*-squared of the ORIV regressions on columns 4 to 6 is calculated as the square of the coefficient.

	OLS			ORIV		
	(1)	(2)	(3)	(4)	(5)	(6)
EA PGI child	0.285***			0.527***		
	(0.015)			(0.021)		
EA PGI mother		0.213***			0.368***	
		(0.015)			(0.021)	
EA PGI father			0.216***			0.402***
			(0.015)			(0.022)
R-squared	0.081	0.045	0.047	0.278	0.135	0.162
N	4,032	4,032	4,032	4,032	4,032	4,032

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Table G23 Results of the OLS and ORIV regressions explaining the standardized offspring Key Stage 4 grade. The R-squared in columns 4 to 6 is equal to the square of the coefficient. Robust standard errors in parenthesis.

G.10 Testing conditional independence

This section tests the independence of the child EA PGI conditional on parental PGI's. Table G24 shows that the existing association between parental educational attainment and the child PGI – between 0.08 and 0.125, column 1 and 3 – jumps to a well-defined zero – columns 2 and 4 –when parental PGI's are added as controls.

	EA PGI child (23andMe)		EA PGI child (UKB)	
	(1)	(2)	(3)	(4)
Father years of education	0.082***	-0.005	0.096***	-0.003
	(0.009)	(0.005)	(0.010)	(0.005)
Mother years of education	0.094***	-0.002	0.125***	-0.005
	(0.010)	(0.005)	(0.010)	(0.005)
EA PGI mother (23andMe)		0.490***		
		(0.007)		
EA PGI father (23andMe)		0.715***		
		(0.008)		
EA PGI mother (UKB)				0.490***
				(0.008)
EA PGI father (UKB)				0.687***
				(0.008)
R-squared	0.076	0.795	0.120	0.794
N	4,032	4,032	4,032	4,032

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Table G24 Results of the OLS regression explaining the EA PGI of the child. Robust standard errors in parenthesis.

H Replicating Dickson

This section replicates the results of Dickson et al. (2016). They exploit a schooling reform in the UK that raised the minimum school leaving age from 15 to 16 years in 1972. Table H25 replicates Table 9 in their paper, for Key Stage 4. The three columns show results using different bandwidths (respectively 3, 6 and 12 years around the threshold). While our sample size is somewhat smaller than in Dickson et al. (2016), we are able to replicate their results fairly well: Table H25 show our reduced-form estimates of the Raising of School Leaving Age (ROSLA) ranging from 15.6 to 25.0, against estimates between 16.4 and 23.5 in the original paper. Table H26 shows the results of the corresponding first stage regressions explaining mother and father years of education. While the coefficient of the reform is always significant for the mother, The F-statistic is only above 10 when the bandwidth around the reform threshold is of 6 or 12 years – columns 2 and 3. The reform is nonetheless a weak instrument for the father education, with the F-statistic being less than 10 for all three bandwidths around the reform.

	(1) Key Stage 4 Score	(2) Key Stage 4 Score	(3) Key Stage 4 Score
RoSLA Treatment of mother (type 1)	19.579*	15.663*	16.866**
	(9.702)	(6.458)	(6.257)
RoSLA Treatment of father (type 1)	25.007*	21.317**	24.958***
	(9.827)	(6.845)	(6.621)
Mother's Age at child's birth (born before sample window)	5.725**	5.443***	4.108***
	(1.785)	(1.194)	(1.027)
Mother's Age at child's birth (born in sample window)	7.267***	6.761***	4.580***
	(1.858)	(1.188)	(0.852)
Mother's Age at child's birth (born after sample window)	7.511***	7.588***	5.014***
	(2.118)	(1.475)	(1.180)
Father's Age at child's birth (born before sample window)	1.723	2.264*	1.840*
	(1.676)	(1.116)	(0.795)
Father's Age at child's birth (born in sample window)	2.470	3.309*	2.673**
	(1.916)	(1.297)	(0.945)
Father's Age at child's birth (born after sample window)	2.415	3.309*	2.439
	(2.184)	(1.626)	(1.333)
R-squared	0.213	0.205	0.191
N	1,353	3,587	6,593
Outcome Mean	433.33	426.89	419.48
Outcome SD	145.17	145.22	147.03
Treatment as % of SD	30.71	25.46	28.45
Treatment joint significance p-value	0.0048	0.0002	0.0000

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Table H25 Results of the OLS (reduced form) predicting Key Stage 4 results.

	Mother years of education			Father years of education		
	(1)	(2)	(3)	(4)	(5)	(6)
RoSLA Treatment of mother (type 1)	0.289*	0.319***	0.263***			
	(0.142)	(0.081)	(0.063)			
RoSLA Treatment of father (type 1)				0.373	0.854**	0.361
				(0.480)	(0.284)	(0.208)
F-statistic	4.129	15.522	17.631	0.602	9.011	3.023
R-squared	0.004	0.006	0.004	0.001	0.004	0.001
N	970	2,523	4,319	970	2,523	4,319

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Table H26 Results of the OLS (first stage) predicting maternal and paternal education.

Next, we replicated the analysis in our analysis sample, where we observe genetic information on both parents and children. Table H27 compares the OLS, ORIV and ROSLA reform for the three bandwidths around the reform. The OLS (columns 1, 4 and 7) replicates our simple intergenerational association. ORIV (columns 2, 5 and 8) replicates our results controlling for parental EA PGIs using ORIV. Columns 3, 6 and 9 show the reduced form effect of the ROSLA reform using the same specification as in Dickson et al. (2016), but using our analysis sample. For the most part, exploiting the ROSLA reform in these smaller subsamples leads to insignificant results. Table H28 depicts the first stage of the ROSLA reform in the same subsamples. The F-statistic is always below 10 for maternal education, whereas for the father, the coefficient even has the opposite sign as would be expected. In short, this analysis simply does not seem viable in our smaller baseline sample.

	Bandwidth 3 years			Bandwidth 6 years			Bandwidth 12 years		
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
RoSLA Treatment of mother (type 1)			12.039			3.683			-1.163
			(13.798)			(9.143)			(9.074)
Mother years of education	13.009***	9.355***		13.860***	7.656***		13.512***	6.968***	
	(3.092)	(2.397)		(1.831)	(1.499)		(1.470)	(1.209)	
RoSLA Treatment of father (type 1)			15.871			8.104			21.305*
			(13.114)			(9.269)			(8.852)
Father years of education	12.556***	9.235***		11.751***	7.170***		11.023***	5.855***	
	(3.048)	(2.316)		(1.829)	(1.384)		(1.419)	(1.092)	
Mother's Age at child's birth (born before sample window)			4.938			4.532**			0.905
			(2.773)			(1.736)			(1.465)
Mother's Age at child's birth (born in sample window)			5.571			5.534**			2.079
			(2.907)			(1.739)			(1.268)
Mother's Age at child's birth (born after sample window)			6.215			6.318**			1.742
			(3.292)			(2.134)			(1.737)
Father's Age at child's birth (born before sample window)			3.484			2.060			2.596*
			(2.353)			(1.624)			(1.087)
Father's Age at child's birth (born in sample window)			3.786			2.975			3.708**
			(2.678)			(1.876)			(1.280)
Father's Age at child's birth (born after sample window)			4.385			2.404			4.109*
			(3.010)			(2.349)			(1.783)
Mother EA PGI (ORIV)		20.794*			31.033***			31.602***	
		(8.238)			(5.072)			(3.892)	
Father EA PGI (ORIV)		21.638**			33.939***			37.559***	
		(8.339)			(4.816)			(3.702)	
R-squared	0.120	-	0.237	0.114	-	0.224	0.097	-	0.188
Obs	591	591	591	1,557	1,557	1,557	2,800	2,800	2,800
Outcome Mean			452.59			451.58			447.88
Outcome SD			129.43			129.27			130.87
Treatment as % of SD			21.56			9.12			15.39
Treatment joint significance p-value			0.3218			0.6014			0.0548

* p<0.05, ** p<0.01, *** p<0.001

Table H27 Results of OLS and ORIV regressions predicting the offspring Key Stage 4 grade.

	Mother years of education			Father years of education		
	(1)	(2)	(3)	(4)	(5)	(6)
RoSLA Treatment of mother (type 1)	0.308	0.290**	0.140			
	(0.186)	(0.100)	(0.073)			
RoSLA Treatment of father (type 1)				-0.369*	-0.206*	-0.285***
				(0.173)	(0.101)	(0.074)
F-statistic	2.744	8.480	3.656	4.520	4.147	14.974
R-squared	0.005	0.005	0.001	0.008	0.003	0.005
N	591	1,557	2,800	591	1,557	2,800

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Table H28 Results of the OLS (first stage) predicting maternal and paternal education.